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A Tribute to Stanley Aronson, MD

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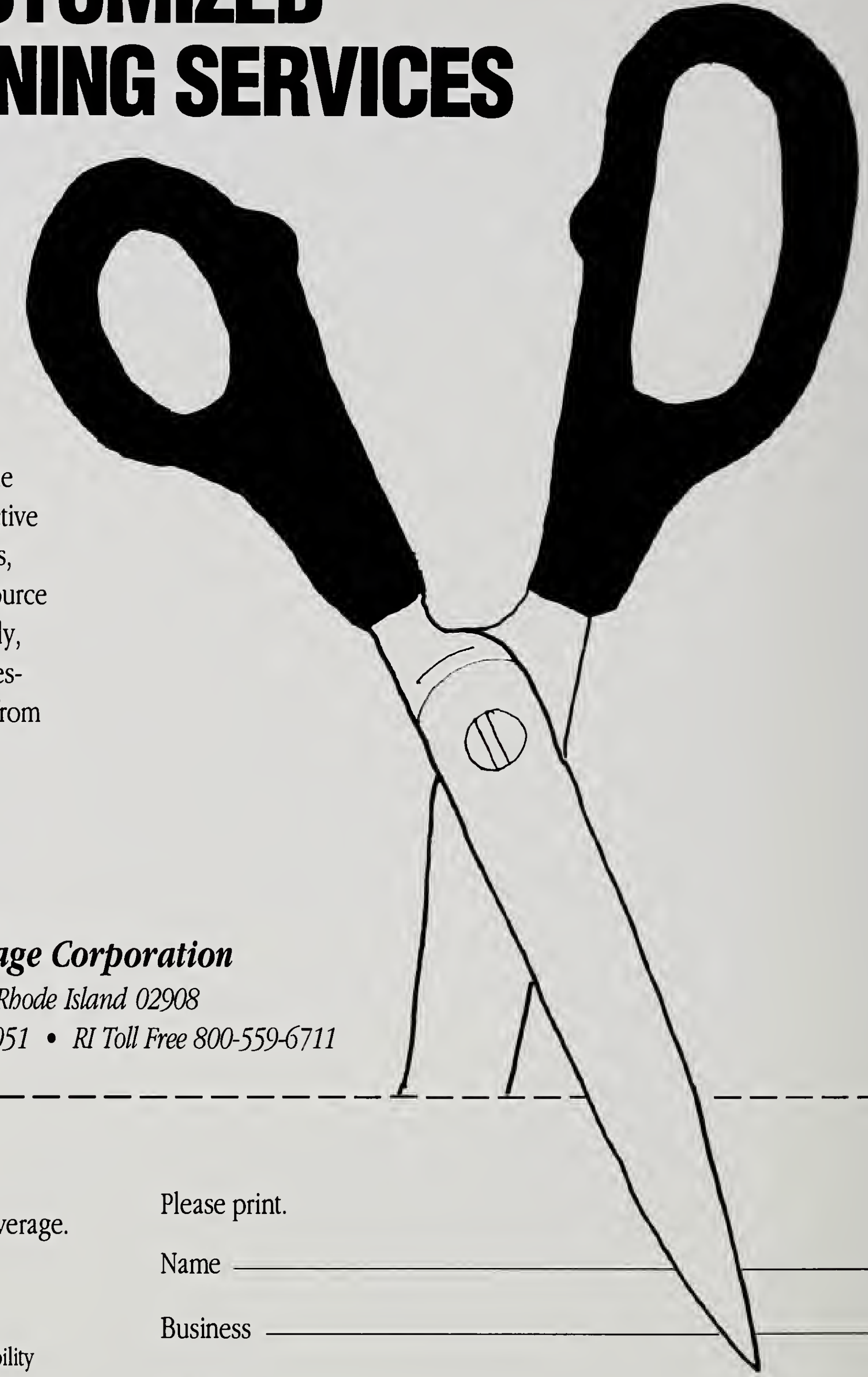

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Cover: *"THE LEARNING TREE,"* watercolor by Claudia Cockerill.

At the entrance to Arnold Lab, the administrative center of the Brown University School of Medicine is the *planatus orientalis*. This tree is the presumed descendant of the tree under which Hippocrates taught and wrote, along with his disciples, the medical commentaries on the natural origins of all diseases.

Dr. Stanley M. Aronson, Brown's founding dean of medicine, donated the *planatus orientalis* to the medical school. Claudia Cockerill, coordinator of the Affinity Group Program and a faculty mentor to the "Art and Medicine" first year affinity group medical students, has exhibited at the Providence Art Club.

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TRIBUTES

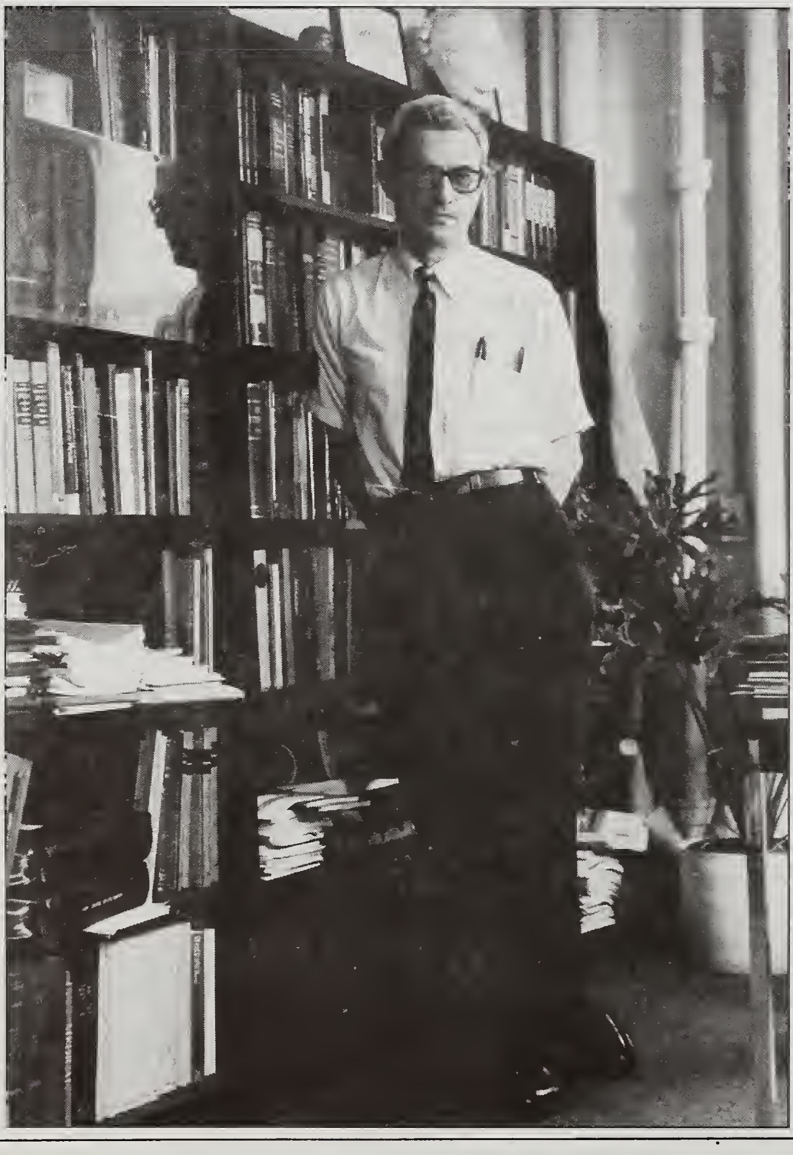
NOTE: Sketches throughout the issue are the work of Stanley M. Aronson, MD, from the program covers created for the Brown University Medical Student chamber music performances.

A Tribute to Stanley M. Aronson, MD

Milton Hamolsky, MD

When I was invited to share in a tribute to Stan Aronson, I accepted promptly - with two certainties and one addendum:

1.) I would fail to meet the desired goal. There is only one individual who could encompass Stan's professional accomplishments and contributions - from neuropathologist to founding dean of Brown University's School of Medicine, to one of medicine's great men of letters, to a national and international consultant and true expert on the spectrum of medical education around the world, to a senior Rhode Island medical statesman on the Board of Medical Licensure and Discipline - and place his stature properly in the sweep of history - and wrap it all in the felicity of the happy phrase. But, of course, he would not do so.



2.) There is no greater privilege than to share in the attempt to honor a colleague and a friend who has, indeed, achieved the pinnacle as one of the truly great men of American medicine. And so, with the skill honed by years of administrative legerdemain I turned to a series of enthusiastically eager (and gracious) participants - each with a special frame of reference to essay the mosaic.

3.) An addendum: Because this issue of tribute to Dr. Aronson was conceived, planned and brought to fruition without any participation by Stan (or, we believe, any awareness), I presume to offer the following: Those who know Stan to some degree - and the perceptive reader of this issue - know that Dr. Stanley Aronson does not stand alone. There has been - and is - no Stan Aronson without his life-long companion and classmate and colleague and friend and true soulmate - Dr. Betty Aronson. If he had been permitted to participate, I know that Stan would have diverted any semblance of tribute to his deeply-loved and respected wife. And so, we offer this shared tribute to each and to both.

Milton Hamolsky, MD, is Professor Emeritus of Medicine, Brown University School of Medicine, and Chief Administrative Officer, Rhode Island Board of Medical Licensure and Discipline.

Continuing an Editorial Tradition of Good Taste

Joseph H. Friedman, MD

When it became public knowledge that I was going to become the editor-in-chief of *Medicine & Health/Rhode Island*, several people asked, in the context of congratulating me, whether I was going to "take over" writing Dr. Aronson's monthly column. Each person looked remarkably happier on hearing that Dr. Aronson was going to continue contributing to the journal without a pause (or editorial oversight, I should point out.) Socrates noted that a wise man knows what he does not know, but Lucille Ball actually put it better, at least for this case. "Knowing what you cannot do is more important than knowing what you can do. In fact, that's good taste."

If anything, I hope to display good taste in the position of editor. I took the position in part to take on a new challenge, but also to follow in Dr. Stanley Aronson's shoes. For an average-sized man, he has enormous shoes that will never be quite filled as he cuts back on some of his enormous range of activities. I moved to Rhode Island after Dr. Aronson had stepped down as Dean and therefore had only a distant relationship with him, but his influence is everywhere. From my colleagues who graduated from Brown in the seventies, to the fruits of his research decades ago (did you know that Dr. Aronson first described Lewy Body disease, now considered the second most common cause of dementia in the west?) to the improved quality of medical practice in the state, his influence is undeniable. To pick up even this one thread of the complex fabric Dr. Aronson has been weaving is a challenge and an honor that I hope I will rise to.

Dr. Aronson was going to continue contributing to the journal without a pause (or editorial oversight).

Joseph H. Friedman, MD, is Chief, Division of Neurology, Memorial Hospital; Professor of Clinical Neurosciences, Brown University School of Medicine; and Editor-in-Chief, Medicine & Health/Rhode Island.

The Contributions to Neuropathology of Dr. Stanley M. Aronson

Daniel P. Perl, MD

In his long career, Stanley M. Aronson has excelled in many different areas – as a pathologist, educator, administrator, dean, essayist, medical ethicist, carpenter, gardener, winemaker, etc. However, the field in which he received his formal training and with which he is most closely associated is that of neuropathology. Stan trained in neuropathology at Columbia University College of Physicians and Surgeons in the early 1950s under Dr. Abner Wolf. The vast majority of his tenure as a practicing neuropathologist was served at the State University of New York, Downstate Medical Center (now the State University of New York Health Science Center at Brooklyn) and its primary hospital unit, the Kings County Hospital. From 1954 to 1970, Stan served as the Director of the Neuropathology Service at Downstate; during that 16 year period he made his major contributions to the field. In this brief essay, I would like to return to those days and review some of what I believe were Stan's unique and lasting contributions to neuropathology, highlighting the three classic components of the life of the academic physician, namely, patient service, research and teaching.

Abbreviations Used:

DLB	dementia with Lewy bodies
JCDH	Jewish Chronic Disease Hospital

PATIENT SERVICE

As the Chief of Neuropathology at Kings County Hospital, Dr. Aronson was ultimately responsible for determining the pathologic diagnoses on all neurosurgical specimens, nerve and muscle biopsies as well as evaluation of all autopsy-derived nervous system specimens. As I recall, the neurosurgical workload was not particularly overwhelming in scale and examples of neoplastic lesions were relatively infrequently encountered. With the introduction of the techniques of enzyme histochemistry into muscle biopsy diagnosis, increasing numbers of requests were made for such evaluations. Stan was quick to recognize the technical difficulties involved in such preparations and encouraged his former trainee and then young Assistant Professor, Dr. Joanna Sher, to develop an expertise in this area. She eagerly took over this aspect of the service and developed one of the first diagnostic muscle biopsy histochemistry laboratories in the United States. Following Stan's encouragement and support, her expertise in muscle biopsy interpretation became widely known and formed a major component of her long and productive career.

Stan concentrated on the post-mortem material, and here he was a master. Working with a series of superb residents and fellows who were attracted to his division, each autopsy case was meticulously described, dissected and sampled, leading to the final detailed neuropathology report. One should remember that the 50s and 60s constituted the latter portion of the golden age of the autopsy. The morgue was still considered a place where the dead had an opportunity to teach the living. Medicine had yet to see the miracle of nervous system imaging achieved by CT and MRI scans, and neurologists and radiologists had not yet acquired the hubris of thinking that any and all pathologic processes present in a patient could be visualized premortem on a computer-generated scan. Furthermore, issues such as cost containment and avoiding malpractice litigation had not yet dampened the enthusiasm for obtaining autopsy permission. During Stan's tenure, Kings County Hospital was one of the largest hospitals in the United States. Using a large number of 40-bed open wards, it attempted to diagnose and treat many of the sickest inhabitants of Brooklyn, New York City's most populous borough. As might be expected, this led to large numbers of in-hospital deaths and with the high autopsy rates of that era, well over 1,000 complete autopsies were performed each year. Indeed, a paper from Kings County reported cumulative data on a consecutive series of 13,000 autopsies performed between 1961 and 1970.

Stan developed a coding system to track the clinical and pathologic findings on each case seen at post-mortem. This system represented the pre-computer era equivalent of a data base file and allowed for the ready retrieval of examples of particular clinical or pathologic entities. This served as an extremely valuable educational resource as well as the framework for the case series that he would incorporate in many of his publications. Members of the Department of Pathology as well as other faculty within the School of Medicine also used this system.

RESEARCH

In his long tenure as a neuropathologist, Stan never functioned as a bench scientist, although early in his career he had carried out research with Gregory Schwartzman (noted for his description of the "Schwartzman reaction") at Mount Sinai Hospital. As a neuropathologist, Stan's research activities mostly involved observations made on post-mortem material, frequently engaging in a form of "post-mortem epidemiology." Clinical and autopsy data derived from large numbers of cases were evaluated and sorted with respect to which types of patients were most likely to develop a variety of neuropathologic lesions. The cases had all been described in detail and were extensively sampled for histologic evaluation. In most years, Stan performed all of the diagnostic sign-outs. The histologic methods employed the standard tools of the day. The hematoxylin and eosin stain served as the primary morphologic tool and, when required, a relatively short list of special stains was employed in order to demonstrate specific lesions, such as, myelin loss, Alzheimer's disease-related changes and stains for the presence of bacterial and fungal organisms. His trainees would be repeatedly amazed at Stan's ability to discern subtle changes in the tissues based on looking at a hematoxylin and eosin stained section. Typically, when special stains were employed it was merely to demonstrate to a doubting resident that a histologic change that Stan had noted on H and E was really present.

During his career, electron microscopy emerged as a valuable morphologic tool, both for research and for diagnosis. EM was available in his laboratory and, although Stan never became proficient in its use, several of his neuropathology fellows and younger colleagues employed the technique. He encouraged his students and trainees to go beyond the methods immediately at hand and, whenever possible, to gain the advantage of new or different approaches or techniques. If this meant a rotation at another medical center, he was very supportive. For example, one of his trainees expressed an interest in lipid chemistry. Stan encouraged an outside hands-on rotation to learn the necessary techniques and then provided funds and equipment to establish a small lipid chemistry laboratory within neuropathology. By the time the powerful investigative tools of immunohistochemistry and molecular biology were developed, Stan was no longer active in diagnostic neuropathology.

Nevertheless, Stan Aronson, using this classic morphologic approach, made a number of significant research contributions in two particular areas, namely, stroke and the sphingolipidoses.

STROKE

As mentioned above, Stan had access to a large number of post-mortem specimens on which he and his fellows had performed detailed neuropathologic evaluations. These were documented in lengthy typewritten neuropathology protocols that were collated into large bound volumes and filled long rows of several sturdy bookcases. In addition, data were collected on the presence or absence of a variety of relevant lesions. At some point, these data were extracted and entered on IBM sorting cards, then the only method for "rapid" retrieval by category. It would be several decades before the advent of affordable computers, spreadsheets and data base managers, all of which would have turned this tedious operation into a much more efficient process. Most of the sorting and categorizing were done by hand and the number of variables for analysis was, by necessity, limited. Nevertheless, by collecting data from thousands of autopsies certain important relationships among the data emerged. These provided important insights into the distribution of a variety of cerebrovascular lesions among certain populations. For example, Stan and his colleagues demonstrated the dramatic tendency for diabetics to develop encephalomalacia, particularly in the form of basal ganglia and pontine lacunar infarcts. Hypertension, in combination with diabetes, showed a strong synergistic effect in producing such lesions. Although these concepts had been previously suspected, it was really Stan's work that definitively established the extent of the connection. Surprisingly, Stan showed that hypertension in diabetics partially protected these patients against the rapidly fatal lesion, primary intracerebral hemorrhage. This was an unexpected result and suggested to Stan that changes in the small caliber arteries and arterioles of the brain that are encountered in the diabetic might prevent their degeneration and ultimate rupture when subjected to sustained hypertension.

Today, with autopsy rates barely reaching 10% of deaths, even in major teaching hospitals, such "epidemiologic" approaches are hardly attempted. However, when Stan Aronson's papers were published, autopsies were performed on more than 50% of in-hospital deaths and therefore represented a relatively complete sampling of such deaths. Although he was meticulous in urging caution related to possible hidden biases, such as variables related to hospital referral patterns, much of Stan's data could be successfully extrapolated. From a current perspective, we are forced to turn to data of this type as they are amongst the only valid population-based observations employing post-mortem observations.

SPHINGOLIPIDOSES

Soon after Dr. Aronson arrived in Brooklyn, he established a close and longstanding friendship and collaboration with Dr. Bruno Volk and his colleagues at the Jewish Chronic Disease Hospital (now the Kingsbrook Jewish Medical Center). The Jewish Chronic Disease Hospital (JCDH) was within blocks of Kings County Hospital and this insured close interaction between the two groups. Dr. Volk was the Director of Laboratories at the JCDH, an institution which provided long-term care to children with hereditary degenerative disorders of all types, in particular, the sphingolipidoses. Those were the days before heterozygote detection, carrier screening campaigns and amniocenteses, and relatively large numbers of children with Tay Sachs disease and allied disorders were referred to the JCDH for terminal care and thus were available for study. Stan published a large series of papers involving studies on the JCDH patients. He reported on a number of morphologic aspects of the lipidoses including descriptions of the brain enlargement (megalencephaly) seen in such patients and other histopathologic features that were unique to long-term survival. He also participated in some of the early attempts at biochemical characterization of such cases..

Dr. Aronson, working with an epidemiologist of Greek extraction, Nikos Myrianthopoulos, began studies on the epidemiology of Tay Sachs disease. Stan organized a family registry for Tay Sachs Disease and identified over 300 families with the disorder. Using data that they had collected, Aronson and Myrianthopoulos estimated that the heterozygote carrier frequency for Tay Sachs disease was between 0.025 and 0.030 among the Ashkenazic Jewish population of the U.S. Following the work of John O'Brien and coworkers, who separated hexoaminidase A and B and provided a reliable means for identifying heterozygote carriers, the true gene carrier frequency was definitively determined. It turned out to be almost exactly what Aronson and Myrianthopoulos had predicted. Stan also characterized differences in the carrier rates betweeen the Ashkenazic and Sephardic Jewish populations, noted an especially high frequency among those originating in Lithuania and adjacent territories, and speculated on how early migration patterns had led to the marked differences encountered centuries later. It was a chance for Stan to incorporate his knowledge and fascination with early Judaic history with medicine.

CLINICOPATHOLOGIC CORRELATIONS

Stan Aronson and his colleagues also provided a number of papers reporting significant clinical and pathologic observations. For example, writing with Haruo Okazaki, and Louis Lipkin, Stan identified two cases of progressive dementia, which were associated with prominent Lewy body formation in the cerebral cortex. These appear to be the first pathologically diagnosed cases of what is now referred to as dementia with Lewy bodies (DLB).

In 1966, Al Spiro of the Albert Einstein College of Medicine, described a 12 year old child with weakness who had a very distinct appearance on muscle biopsy. He called the disease myotubular myopathy, feeling that the muscle fibers present had failed to properly develop and retained their fetal myotubular appearance. Some months later, Stan, with Joanna Sher as the first author, published the findings of two sisters who showed an almost identical pattern on muscle biopsy. They correctly recognized that their family and Spiro's patient likely suffered from the same illness. However, they remained unconvinced that these were myotubular cells and coined the term "centronuclear myopathy". The nature of the disease became an issue for discussion in neuropathology circles and it was ultimately the consensus that the changes noted were not consistent with myotubular arrest. Since then, the preferred term for the disease has been centronuclear myopathy.

I have always been impressed that for many years Stan ran a weekly clinico-neuropathologic conference with the neurology and neurosurgery staffs. Many of these conferences were published in the form of a monthly feature of the journal *Diseases of the Nervous System.*. These sessions certainly served as an important basis for the teaching of a generation of neurology, neurosurgery and pathology residents who took their training at Kings County Hospital. Over the years the cases spanned the entire range of nervous system disorders, including infectious, degenerative, neoplastic, metabolic diseases, etc. I was constantly impressed at how this series would continue to come up with one fascinating case after another. It reflected the excellent material then being seen on the autopsy service at Kings County. It is unfortunate that this material was not collected and reprinted as a separate volume since the cases were all well described clinically and pathologically and could have served as a valuable teaching reference to a much broader community.

TEACHING

Above all, Stan Aronson, the neuropathologist, was the master teacher. Each year he would deliver a series of didactic lectures on neuropathology to the 2nd year medical students. This began at Downstate and continued at Brown. His lectures were known for their clarity and for the way in which he wove clinically relevant material into the basic pathologic concepts. But most of all, it was Stan's eloquent lecturing style (what someone has referred to as his "Shakesperian" delivery) that made them truly unique. Although his lectures were given without notes, no word or phrase ever appeared to be out of place or awkward to the ear. It was as if the lines had been polished and rewritten by a team of English professors and literary editors prior to being uttered. His lectures were laced with frequent literary references and analogies. Some of these references were remarkably arcane, reflecting Stan's broad literary background. Such references frequently inspired inquisitive students to seek out their source. Almost invariably upon reflection one would find that the reference, no matter how obscure, was precisely appropriate for the point being made. I am not sure how well such references would fare with today's generation of medical students raised on sitcoms and MTV but in those days many found the literary references almost as fascinating as the subject matter.

I find remarkable the large number of the Downstate medical students who were attracted into the field of neuropathology following being exposed to Stan's 2nd year neuropathology lectures. Many have gone on to leading positions in this discipline. Included among his former Downstate students from that era are Marius Valsamis (who practiced neuropathology at NIH, Albert Einstein and New York Medical College), Lester Adelman (Tufts), Nancy Peress (SUNY Stony Brook), Suzanne Mirra (formerly at Emory and now recently appointed Chairman of Pathology at Downstate) and myself. I am not aware of any other neuropathologist who personally inspired this number of medical students to go into this rather small subspecialty. To his further credit, virtually all of these individuals went on to be honored by their respective schools for their superior teaching abilities. I doubt if any of us could give a lecture in a style that is in any way similar to Stan's; I cannot. Stan never consciously attempted to train any of us in how to deliver a lecture. Yet somehow, presumably by example, something was transmitted that enabled virtually every one of Stan's former students to also become an inspiring teacher. He set the example and by stimulating his students he made an important and lasting legacy to the field of neuropathology.

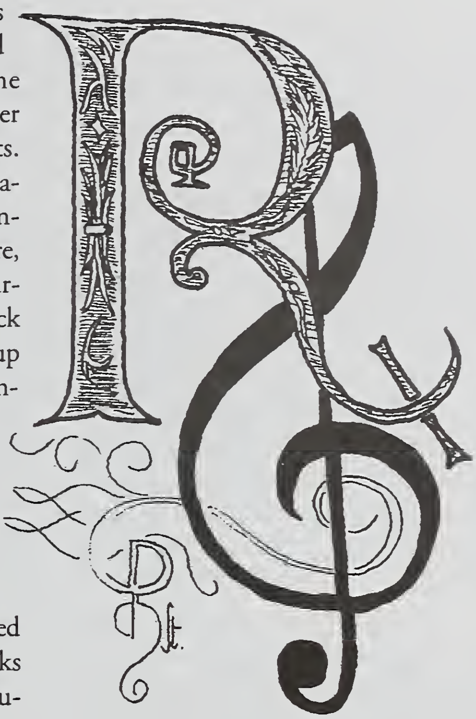
In addition, at Downstate Stan established a neuropathology training program that was, over the years, highly thought of and was very productive. Not only were the above mentioned Downstate students attracted to train with Stan in neuropathology, a number of other talented individuals were also attracted by Stan to Downstate for their neuropathology training. These include Haruo Okazaki (Mayo Clinic), Juan Olvera (Mexico City), Julio Garcia (Henry Ford Hospital, Detroit), Joanna Sher (SUNY Downstate), Archinto Anzil (SUNY Downstate). Each went on to make important contributions to neuropathology.

Stan formed close attachments to his fellows. When it came time for them to move on to independent faculty positions, he provided guidance and strong support. Because Stan was well known and highly regarded in pathology and neuropathology circles, his support of a candidate for a position carried great weight. For example, Nancy Peress told me that at the completion of her fellowship, Stan arranged a job interview for her with Dr. Marvin Kushner, then Dean of the School of Medicine and Chairman of Pathology at SUNY Stony Brook. Dr. Peress arrived at the interview in the latter stages of pregnancy with her second child. With her superior qualifications and Stan's enthusiastic endorsement, on their first meeting Dr. Kushner indicated that he would be "delighted" to hire her.

Over the years, Stan's trainees repeatedly turned to him for both professional and personal advice. This was always given following thoughtful reflection and consideration of what was best for the individual seeking his counsel. In my own case, at a point where I had to make a difficult career decision, Stan offered advice that differed from what I thought was the best decision. He carefully explained his reasoning, and I ended up taking his advice. Several years later it was clear that he had been correct. I continue to feel fortunate that he saved me from becoming involved in a difficult, if not impossible, task. He closely followed his trainees' lives and careers and was available whenever any needed his help.

Every year at the American Association of Neuropathologists' annual meeting, virtually all of the practicing neuropathologists gather both to present papers describing their latest work and to socialize. Stan had served as Secretary-Treasurer and President of this organization, but as Dean at Brown he decided that he no longer had time to regularly attend the meetings. About 15 years ago, Stan decided that he would return to the neuropathology meeting as an opportunity to see some old colleagues and friends and get together with his former students and fellows. Deciding that a dinner was the most appropriate venue for this reunion, we invited all of his former students and trainees to the back room of a rather gaudily painted but excellent Italian restaurant. I remember a very warm evening with much good food and abundant wine followed by a number of speeches. These included stories of the days in training in Brooklyn as well as many acknowledging Stan's impact on our collective careers and lives.

The following year Stan did not attend the meeting but his fellows nevertheless decided that we would get together for a dinner without him. An appropriate restaurant was quickly found and the session was well attended. A tradition had begun and I can report that every year since then, Stan's boys (and girls) have met for a dinner in his honor. By now, this annual dinner not only welcomes Stan's trainees, but also includes his so-called "trainee grandchildren" (that is, those trained by his trainees). As might be expected, the event has grown large. For me, this dinner represents a particularly vivid demonstration of Stan's most lasting contribution to the field of neuropathology. He attracted to the field and trained a substantial number of today's leading neuropathologists. They, in turn, have carried on his tradition of excellence and have themselves trained others who, in the future, will continue to practice and teach further students. Each year I am struck by what a talented and varied group they are and how much they have contributed to their institutions and to their areas of expertise. This is clearly Stan's most significant legacy to his profession.



There is not a single paper in neuropathology for which Stanley Aronson is not known. His published legacy is a body of work which marks the end of the era of descriptive neuropathology. But even more importantly, his legacy is in the people he trained.

In neuropathology, as in the rest of his life, Stan Aronson is remarkably knowledgeable in a wide variety of subjects. Given some prompting, Stan can and will talk with eloquence and understanding on gardening, building stone walls, the ancient migration of the Jews, and in the same manner on stroke, brain tumors and HIV infection.

For many years, Stan served on the Medical Advisory Board of the National Multiple Sclerosis Society. This always seemed odd to me since he had never actively done research on the condition. Nevertheless, when you spoke to him about the disease and the state of our understanding about it, he was knowledgeable and insightful about where promising research leads might emerge. He obviously brought to the discussions at the MS board a breadth of knowledge and perspective that was valuable and not confined to the boundaries of expertise in any narrow technical discipline. For me this was and is one of Stan's most valuable skills. That is, his ability to weave a breadth of knowledge which crosses so many diverse areas. Today we see too many "experts" whose understanding focuses narrowly on either a single technique or discipline. They fail to possess a fund of knowledge that allows them to extend this expertise beyond their formal training. For example, I meet many molecular biologists who are knowledgeable on all of the latest tricks of how to dissect out and study the details of the genome. Yet, they know virtually nothing of biology and disease and haven't a clue as to how to interpret their findings in the context of a living organism or a pathologic state. Stan showed us that you have to be more than a well-trained neuropathologist and he challenged us to continually broaden our horizons.

I do hope that Stan Aronson will find a way to continue to provide his insights into neuropathology specifically and life in general. I congratulate him on his well-earned retirement from being editor of *Medicine & Health/Rhode Island* and acknowledge that we still have much to learn from him.

ACKNOWLEDGEMENT:

I would like to thank Drs. Lester Adelman and Nancy Peress for sharing their reminiscences and helpful advice in the preparation of this document.

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A Dean for All Students

Edwin L. Zalneraitis, MD

When invited to write about Dr. Stanley Aronson's role as a Dean and mentor for Brown University medical students, I eagerly agreed to accept the offer. I did so, not only because he was the most important leading influence and mentor in my career, but also because I have come to know how important he has been to the personal and professional lives of a great number of Brown Medical School students over the years.

Although frequently paraphrased and quoted, but I suspect not very often read, Charles Coleb Colton (1780-1832) wrote in *Lacon*, vol. Ib. no.217: "Imitation is the sincerest of flattery." While I am not certain that I am talented enough to truly imitate Dr. Aronson's writing style, I will make something of an attempt as a tribute to him. I have selected a title to play on the phrase "A man for all seasons." The latter is a title for a passage composed by Robert Whittinton (c.1480-c.1530) for school boys: "Time requires a man of many abilities and moods, marvelous mirth and sad gravity." Being an inaugural Dean of the School of Medicine, and a mentor to so many students over such a long period of time, requires a man of many abilities and dedication. Indeed Dr. Aronson's abilities as a physician, scientist, writer, educator, philosopher, ethicist, husband and parent - - made him uniquely qualified for the role he was to play for us as students. His dedication is what has made his application of these talents so special.

As for the title "Dean," it has origins in both Greek and Latin. To understand its meaning better, the Oxford English Dictionary is very helpful. The word has astrologic derivation in both Greek and Latin, referring to the ten degrees of the zodiac sign and applied to a commander of ten. Hence, over the centuries, it has been applied in situations such as a commander of a division of ten, or the head of ten monks in a monastery. Eventually, from its monastic use, it came to refer to the head of a chapter or body of collegiate or cathedral church. The word went through a series of more generalized ecclesiastical applications, indicating the head of various church organizations, before it became used in the colleges of Oxford and Cambridge. "Dean" became the title given to one or more resident fellows appointed to supervise the conduct and studies of junior members and to maintain discipline among them. Finally the word was applied to president of a faculty or department of study in the ancient Scotch Universities, and then the modern universities of London and other schools in the British empire.

Dr. Aronson brought leadership and order to the Brown University School of Medicine, and led us to the modern school as it is today. For students, he was a constant presence in ensuring our education. As a role model and a leader, he commanded our dedication and discipline as we passed through a demanding and thoughtful curriculum. As his students, we all sensed his dedication, and his sincere and direct interest in all of our individual and collective success. We were compelled to achieve at the very highest level possible, in order that we could meet the promise of our Dean, and return his commitment to us. In this sense, Dr. Aronson met the challenge of the tradition of deanship as it evolved in the modern meaning of the word. However, his commitment to his students did not end at the classroom door or with the passing of a diploma into our hands, and it is in this part of his interactions with us that we must consider the other word I was asked to write about: Dr. Aronson's role as mentor to students.

The origins of the word mentor can be found in *Origins: A Short Etymological Dictionary of Modern English* by Eric Partridge (Greenwich House, New York, 1983:.614). Partridge tells us that mentor is "from the root men- (:mon-) to remember. think, counsel." It has apparently evolved from Greek usage of the poet who uses the word mentor as, "the name for the Ithacan noble, whose disguise Athena assumed in order to act as the guide and advisor to Telemachus." Hence as a common noun: "an experienced and trusted counselor." This use of the title mentor is further noted to have been applied by Homer, when he used the name Mentor for the friend of Odysseus who served as the wise counselor in Homer's epic poem. We shall have no difficulty applying the word mentor, as derived from these Greek origins, to our wise and trusted guide, Dr. Stanley Aronson.

Dr. Aronson brought to his role as our mentor, not only the needed dedication and leadership, but also a wealth of experience and wisdom that made him "trusted counselor" to so many of us. While we were students, he would always find time for us to discuss the scientific, the medical, the social, the political, the ethical and even the intensely personal in a wise and supportive way. He could be stern when required, and warm and caring as needed. Most importantly he was never too busy for, or indifferent to, the problems and needs we presented to him. He was ever present in promoting the launching of our careers into training programs after our medical school experience, and remarkably enough, remained interested and involved in a great many of our lives and careers beyond.

Speaking from a personal point of view, it was very comforting to me as I received a steady stream of correspondence from Stan, as he inquired of my progress and provided me with advice at each turn in the road. The value of an experienced and caring mentor, as one makes the difficult career and life decisions, is absolutely invaluable. It is warmly reassuring to pick up the telephone and still be able to reach a friend and mentor who is just as concerned twenty-four years later as he was the day we started down our medical school path. It is stunning to pick up the mail and find a note from a busy scholar, who has the thoughtfulness to take the time to pause and write to inquire about your progress in life and career. Even more remarkable is to find a confidant and teacher who brings life lessons to your problems in a fresh perspective each time, never afraid to ask where else on the page might we color beside between the lines. He has pointed the way without ever expecting that his advice was contingent on our compliance. He has shared disappointments with encouragement, and success with triumphant celebration. For all this, I am truly grateful.

While my personal life was influenced so profoundly by Dr. Aronson, I found it even more remarkable that he has played the role of mentor to so many other students that he has shepherded over the years. He and Betty have made time to come and visit at least yearly wherever I have been, as a teacher to my students, and for personal visits. During these visits he would often ask about other Brown students in the area, and give me news about classmates and students from subsequent years. It became clear that he had continued to reach out and correspond with many of us; and that he made the same effort for them, whenever it was asked of him, as he had for me. Therefore, it is not surprising that, whenever I meet Brown Medical School graduates, most of them share the common experience of his mentoring. This investment of time, effort and caring is a remarkable model for us all to emulate. In fact, I find myself compelled to try to maintain correspondence and contact with many of my own students and residents from the years gone by, and I do so with memories of my relationship to Stan.

Though I trust that Medic*ine & Health/Rhode Island* is passing into very capable hands, we will miss Dr. Aronson's guiding influence. I am sure we all wish him well in his future endeavors (and knowing Stan, there will be more to come that will amaze us all), and look forward to some future contributions to make us all pause for thought, and become a little wiser in the process.

Edwin L. Zalneraitis, MD, is Professor of Pediatrics and Neurology, Pediatric Residency Program Director, and Asisstant Dean for Clinical Education, at the University of Connecticut School of Medicine.

Reflections of a Few Alumni(æ) on a Beloved Teacher

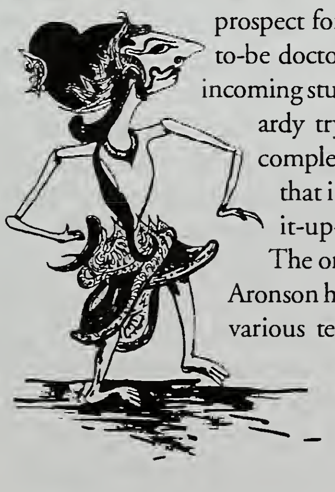
Ruth Sauber

For nine years, I worked as Medical Student Affairs Officer for Brown University's Program in Medicine, at the side of the founding dean, Stanley M. Aronson.

I was in a unique position to see at close hand how Dean Aronson worked his magic on wide-eyed, naïve, incoming medical students, and, during their four years at Brown, helped turn them into caring physicians.

As role model, mentor, and empathetic father figure, he epitomized the finest characteristics of the consummate teacher/practitioner.

Medical school, at best, is a daunting if not frightening prospect for the newly accepted hoping-to-be doctors. But at Brown, these early incoming students had to face double jeopardy trying to adjust to a new and complex learning regimen, and to do that in a brand new, almost "make-it-up-as-you-go" medical school. The original slate was clean as Dean Aronson helped create partnerships with various teaching hospitals, helped develop the curriculum, organized the teaching faculty, operating committees, the administrative staff and developed workable procedures. Regardless of whatever else was going on, he always found time for students. After all, they were the reason that the institution was created in the first place. His office was always open, for whatever was bothering them, however he could help. Just come in and talk, no appointment necessary.



It's one thing to hear about it from me, but let some of the over 600 students he nurtured speak for themselves. Here is what a few of them have to say:

Julianne Ip, MD'78

When I think of Dean Aronson, I think of dedication, of strength and of commitment. Indeed, there is still a sense of awe of his position and of what he represents, the noble profession of medicine.

In the fall of 1975, the ancillary health care workers at the Institutes of Mental Health (the IMH) were going on strike. Dean Aronson announced that he needed us to help... us, the first year medical students... We were reminded of our commitment to helping others... It was a frightening experience and we were unprepared. We were actually helpful and there was an overwhelming sense of satisfaction. We were part of the noble profession of medical caregivers and Dean Aronson.... .gave us the strength of his faith in us. He had high expectations of each of us and accepted nothing less. We hope he is proud.

Thank you Dean Aronson from all the physicians who graduated under your tutelage and guidance.

Preston C. Calvert, MD '79

... Dean Aronson was one of the strongest inspirations in my developing career. He used a gentle sense of humor in dealing with potentially difficult interactions. I will always remember his gentle, but firm admonition to "Be well."

... There was a sense of family and warmth in the Dean's office, fostered by Dean Aronson's own actions and those of his staff... We were all embarked on an adventure of developing and shaping a new medical school and we could not have had a more charismatic and caring leader than Stanley M. Aronson, MD.

Mitchell H. Driesman, MD '77

... To the early students, he was our "father figure." With his thoughtful eloquence, his work ethic, his boundless love of all learning, he was our role model. ... When we had a problem, we brought it to the Dean. When there were celebrations, we shared them with the Dean and his family and he made us a member of his family. ... I still cherish the relationship he fostered and the memories that have endured.

Dean Aronson, thank you and congratulations.

David D. Diamond, MD '78

I was a student in the MMS program being considered for admission to the MD Class of 1977 when I went to speak to Dean Aronson about the possibility of taking a year off to study and travel in Europe ... I wanted my admission deferred ... Dean Aronson sat me down in his office and asked if I had considered if the value of what I planned to do could be favorably compared to the value of a year as a practicing physician.

After a few moments I said that I would rather make the comparison of my whole career enriched by my proposed extracurricular experience with a career without having had such an opportunity He paused, and made one of those Aronson smiles, and I knew then he would agree. I did go to Switzerland and had a wonderful year, and I applied lessons learned abroad in my professional practice and personal life, but I have also benefitted from the perspective I was given that afternoon, and on many other occasions during our mutual tenure at Brown...

Alan T. Kaell, MD '78

I would like to take this opportunity to thank you for your compassion and support. ... It is only through your encouragement and dedication that I was able to complete my MD degree and remain in Providence until 1978.

... Whenever I struggle with forever changing issues of managed care and the current health care crises, I often think of how you would handle the situation ... I am forever grateful to you for instilling your humanitarian values into me.

Paul J. Agatiello, MD '81

I have always felt strongly that Dr. Aronson exemplifies those qualities which are so rare, but are so much in demand to practice committed medicine - humanness, love of life and caring of other people.

David Brooks, MD '76

In the fall of 1972 I arrived in Providence with a wife and a 1 year-old child and the ludicrous dream of becoming a doctor. I had graduated from college in 1968 with a somewhat less than distuinguished record and had immediately gone into the Navy for 3 years ... Brown and Dr. Aronson offered me the opportunity to try my hand as a special student in the Masters in Medical Science, with no promise whatsoever of anything beyond that ... as luck would have it, one of the members of the class of 1976 was detailed in Hong Kong...and through intense wheedling of Dr. Aronson's staff...I managed to take the empty spot.

Thereafter, the 4 years at Brown were among the happiest and most fulfilling of my life. Dr. Aronson had put together a remarkable student body ... leavened with a number of older students such as myself... For the next 4 years our lives revolved around Dr. Aronson's vision of what constituted a proper medical education.

Harold K. Gever, MD '78

... by instinct, I made it a point to mostly avoid a Dean ... Dean Aronson's inviting manner and willingness to take personal interest showed me the side of a Dean which few people commonly see ... the "kinder, gentler" Dean ... I visited his office to seek help in finding a breast cancer specialist for my dying aunt. . . He immediately picked up the phone and called a marvelous specialist in Manhattan and made an appointment for her on the spot. . .the hope and support, which his gesture provided, left an indelible impression of kindness.

Thank you, Dr. Aronson for showing me what a Dean actually is.

Roslyn Chosak, MD '77

During my medical training, Stanley Aronson became a great friend, mentor and colleague. My children recall fondly the wonderful class parties at the Aronson family farm in Rehoboth. And so, for providing me with your mentorship, support and leadership, thank you Dr. Aronson.

Paul vonOeyen, MD 75

Dr. Stanley Aronson has left an indelible imprint on me... From his erudite professional "Mechanisms of Disease" lectures to his weekly "hand-holding" sessions through the first clerkships as Dean.

... Dean Aronson, we salute you as an academician of unsurpassed excellence, a professor of remarkable pedagogical influence for all of us to strive for the highest possible potential, and a truly outstanding professional role model of personal integrity and selfless concern for the betterment of humanity.

Carole St. Pierre-Engles, MD '76

To a first-year student in pathology, he appeared as a tall gray man, very dignified and somewhat intimidating. Yet even that year, he brought this seemingly dry subject matter to life with his presentation. Later, in neuropathology, I found his teaching gift even more impressive. Dr. Aronson captivated us by the manner in which he built a tale, leading us through the details of discovery ultimately to a final revelation. It was not unlike reading a good mystery ... His were lectures I never wanted to miss.

As Dean, my fellow students and I provided him with many challenges. Yet whatever the issue, and however close we were to outrageous, he always listened. Even when situations were rather tenuous, he was able to facilitate compromise. When more significant support was needed, he was there to provide it, and it was invaluable. . .

I am most grateful for having been able to share the universe with Stanley Aronson, MD – teacher, leader, and friend.

Jonathan Gell, MD '75

Dr. Aronson taught us that clinical and even administrative work was a challenge, not a burden. Medicine is the most scientific of the humanities, the most humane of the sciences. This was a core philosophy of Brown University and Dr. Aronson exemplified that tradition. Thank you for the opportunity to remember how important he has been in my career.

John Sheppard, MD'78

Dr. Stanley Aronson stands out in my memory as one of the strongest, yet graciously quiet personalities at Brown. I will forever thank him for encouraging our musical and athletic endeavors, however eccentric, making us understand that diversity is welcome in medicine. He was a great supporter of our student concerts, always attending and sitting in the front row. I felt as if I was performing for him since he sincerely enjoyed classical music. His Multiple Sclerosis lecture in the dreaded B13 dungeon remains indelibly in mind as one of the most intriguing of my 7 years in Providence.

Mark E. Haffenreffer, MD 77

My class had the feeling that Aronson was in charge and expected a lot out of us but that he did allow us room to maneuver.

Stanley Aronson was a true gentleman and a great researcher ... I think that my time with him as Dean of the Medical School was very positive and that I was very fortunate he was involved in education at a time when I needed him most.

Peter A. LeWitt, M.D. '75

One recollection is the May, 1975 issue of the *Rhode Island Medical Journal.* Dr. Aronson arranged for this special issue to serve as the yearbook for the Class of '75, for which we wrote several articles on the development of the Medical School and its first voyagers into the world of medicine. His words ... show his sincere and humane desire to create physicians with the highest qualities that, of course, mirrored the values and capabilities he has so well exemplified. I certainly felt his strong guidance and warmth in teaching and leadership during my education at Brown....

H. Wayne Carver '74, MD '77
Deborah DeHertogh '74, MD '77

Our lives have intersected Dr. Aronson's over the course of 25 years - as our Dean, as counselor for two young medical students contemplating marriage, and as our friend and colleague when we became established in our own careers. For both of us, Dr. Aronson embodies all that a physician should be as a healer, teacher and scholar, and, above all, as a caring human being. We hope, at least and at best, to pass on some of what he taught us to our students and to our children.

Michael Migliori, MD '82

Dean Aronson was taller than anyone in our class, and with his deep voice and eloquent parlance, he was able to command the attention of our rather unique and unruly group. What began as a feeling of intimidation quickly turned to one of admiration and respect.

He showed us what a gentleman could accomplish, and how a physician should act. He was and still is a role model to me. My biggest disappointment in medical school was that Dean Aronson retired before I graduated and wasn't the one who handed me my diploma.

Claudia Gruss, MD '77

His office door was open to us to discuss our most trivial of problems or complaints. He would treat each individual as if his or her concern was the most important matter of the day. When a student was in trouble or ill, he would be there for him. It meant a lot to me when, awakening from emergency surgery, his was the first familiar face I saw ...

He showed us, by example and by his personal support of many different activities, that a good doctor should be a complete individual, not a technician.

... he taught his students the importance of keeping empathy and compassion in the practice of medicine.

John P. Femino, MD '76

I was asked by some fellow students to assist in putting together a yearbook ... I was quite skeptical about this endeavor as much of my photography efforts were frustrated by editors who would require some formatting or editorial slant that would often insert the school messages or agenda between myself as producer and the student as subject. I went to you expecting to struggle for justification, funding and control, and I was amazed at your support and respect. You found the funding somehow and gave me complete control of the project despite its rather unconventional format. Your editorial role was that of nurture, respect and guidance and never did I sense that your agenda restrained me. . .You practiced respect on the job as Dean, and listened to my perspectives and experiences as student - modeling for me the role of physician and patient.

Joel Shalowitz, MD '77

I had the pleasure of having Stan Aronson not only as a Dean but also as a classroom teacher and personal mentor. As Dean, he steered our School from its beginnings to national prominence. In the classroom, he taught us not only neuropathology but also the importance of being a well-rounded human being – with interests in the arts as well as the sciences. I had the opportunity to work with him on an elective rotation in administrative medicine. The experience stimulated my desire and interest to pursue more formal training in that area. … He gave new meaning to the phrase *in loco parentis.*

Anthony Caldamone, MD '75

He met with our class, the first MD class (1975) one Tuesday night in the basement of the Biomed building. He talked in broad terms of his vision for the development of the medical school and in particular, this "first class of Brown medical students." We were all reserved about his presentation until Dr. Aronson said something that was out of the character that we had thought we were dealing with. He turned to the blackboard and wrote down a phone number and said, "This is my home phone number, call it anytime, day or night, if you have a problem or if you just need to talk." … He continues to provide guidance and inspiration through his work ethic, professionalism, sincerity, and dedication to his students long after that 1975 graduating of the first Brown Medical School class.

Mark S. Blumenkranz, MD '75

Dr. Aronson, himself a neuropathologist and a fine scientist rather than a practicing clinician involved with the daily care of patients, nonetheless came to represent the … human and personal side of medicine. The furrow in his brow accentuated by his hand cupped around his chin while reflecting; the slow and thoughtful responses to questions; the dry wit; and most importantly; the obvious concern with which he viewed our early struggles was a source of comfort and reassurance during the early years of the School of Medicine … much of the character of the School … the direction the School took in the first ten years after its founding, is related to his direct and indirect impact. He accomplished this through role modeling, selection of new faculty, and moral and intellectual values imparted to the students, not only as a class, but on a very personal and human level, one on one.

Margaret M. Parker, MD '77

I first met Dr. Aronson when I was requesting to transfer into the second year class of the Program in Medicine so that my husband and I could get married. The request came in May, past the time that the Admissions Committee had met, so the decision rested entirely with Dr. Aronson. As Bob and I have now been married for 24 years, I am especially grateful to Stan for accepting my transfer.

Dr. Aronson's articulate lectures, delivered in his distinctive style, in that basement room with no windows and blue carpeting on the walls, remain among the strongest of my memories of medical school His wisdom and insight, coupled with his fatherly guidance, helped to launch the careers of many aspiring physicians.

James Revkin, MD '81

It is the memories of personal interactions with Dr. Aronson that have left a lasting impression on me. I learned from Dr. Aronson that the capacity to listen … and listen for as long as it takes … builds bonds with people. In the case of patients, being a good listener can help heal them emotionally, if not always physically. The challenge for physicians today, in the face of the pressures of managed care and the bottom line, is to maintain that sense of humanity that Dr. Aronson embodies so fully.

Dr. Aronson, as a physician/educator/listener, expresses the true spirit of what students of the Greek philosophers call the "maieutic method." Interestingly, *maieutikos* is a Greek word with a medical connotation, meaning midwifery, or giving birth. One must be humble enough to listen to one's patients and to other health care providers again and again, to develop and bring forth the "right" treatment plan.

Seth Berkley, MD '81

For me and for many others, he has been a valued mentor and more important, a friend.

Dr. Stanley Aronson is an extraordinary individual. He has touched physicians and patients locally, nationally and around the world. He has done more to improve Rhode Island medicine than any individual in this century. . . He embodies what we strive for in a physician, the highest ethical standards, compassion and empathy for his patients, concern for the poor and commitment to serving their needs, unwavering commitment to medical education, and true scholarship including the ability to communicate his work not only to his peers but to the general public.

Michael Chang, MD '81

He was a source of inspiration and encouragement and personally represented, more than anyone, what was special about the Brown Program in Medicine … that lofty ideal of a humanistic renaissance physician, integrating the very best of both the art and science of medicine.

My last memory of Dean Aronson was at Commencement, when I ran into him amidst all the empty chairs on the Green. I told him that I wished I had my camera with me so I could have a picture of the two of us in our gowns. His response was, "Don't worry, Mike, you'll always remember this in your mind." … Classic Stan Aronson. And you know, I always have.

Josef Machac, MD 78

In those early exciting and tumultuous years of the program, everything had to be invented from scratch, and continuously reinvented, and therefore bore Dr. Aronson's creative stamp. I admire now, as I did then, his patience, steadfastness and ability to listen. I am sure that at least half the time, we were exasperating, but Dr. Aronson was willing to listen and compromise when necessary. Most important, he cared. On an individual basis, he was an advisor and mentor to each one of us. I feel privileged to have participated in the exciting beginnings of the Brown Medical school, and that Dean Aronson was there to guide it. I am grateful for his work for the school and for me personally.

Carl Geyer, MD '80

Dr. Aronson was a giant in my life during the late 70s at Brown. If a lecturer missed a conference due to snow, a late case, etc., Stanley would always fill in with yet another perfect lecture on hemachromatosis or some allied subject that at the time seemed brilliant, lucid and always insightful.

Every fall the leaves turn color and I am reminded of one bright, sunny autumn afternoon full of reds and oranges, and Stanley Aronson walking with slide carousels, ready to lecture yet another class about medicine and, more importantly, about life.

Arnold Rabson, MD '80

I feel tremendously fortunate to have been a medical student at Brown during Dr. Aronson's tenure as Dean. His own day-to-day life, as mentor, teacher, Dean, and often friend, personified his commitment in every way. When students had problems with courses, their health, or even faced personal tragedies, Dr. Aronson was a calming and reassuring presence, who cared deeply about our individual growth and success. Difficulties with courses or clerkships were addressed in a mature and reasoned way, respecting our opinions, instead of dictating to us. When students were sick, Dr. Aronson was more than likely to personally call and see if they needed anything.

Dr. Aronson combined a practical demonstration of the centrality of research and intellectual achievement in the science of medicine with a reverence for patient care and respect for the individual. As Dean, Dr. Aronson planned, shaped, guided, cajoled, persuaded, and even fought when necessary for his vision of the special medical school that Brown became. All of these different activities were accomplished by Dr. Aronson with an unfailing sense of good humor and spirit, even in the most serious of moments. But it will probably be the less serious moments that I remember the longest and with the most affection. Who among us could forget Dr. Aronson's good humor upon the receipt of his annual award at the medical student Senior Show? I can't help but wonder where the "Stanley M. Aronson Endowed Chair of Medicine" is today.

Arthur Horwich, MD '75

As the helmsman of our class and school, Dean Aronson brought so many wonderful qualities to the inaugural voyage: patience with the rigging in its first time out; warmth that glued the "crew" together; a personal fiber of greatness that was so visible and assuring, both in his past accomplishments but, most significantly, in his vision for where this ship was going. As with the *Rhode Island Medicine and Health Journal*, so with us students, you set us on the right course.

Elizabeth George, MD '76

I was somewhat intimidated when in the fall of my 2nd year, Dr. Aronson asked me to meet him for coffee at one of the Thayer Street coffee shops (uh oh - what did I do?). I was a little puzzled when over coffee he unfolded a sketch of a stethoscope shaped into a G clef. Then, the neat plan unfolded of wanting to support students in their talents beyond their medical training - such as music - which made them more well rounded in their lives and in their attitudes. He was keenly aware that encouraging fullness in our lives would have a positive impact on our approach towards patients. Thus, the Brown University School of Medicine Chamber Concerts came into being with concerts by students and faculty in the Crystal Room with wine and cheese receptions afterward. Dr. Aronson's only specification was that he be allowed to design the program cover for each event. [Many are shown throughout this issue.] What I appreciated was the support to continue my flute playing and performance amidst busy medical school days - and I've continued that enjoyment through busy years in Family Practice.

Wilma Schiller, MD '79

Stanley Aronson was remarkable for paying attention to us as people. He was always accessible. He never trivialized our concerns, even if they were, in fact, trivial ...

He cared about his medical students as whole human beings. By his example, he taught us to care about our patients the same way. That is one of the qualities that distinguishes the Brown Medical School and its graduates.

Edward W. Martin, MD '79

While I was a medical student, Dr. Aronson was the primary force behind the development of a Hospice program in Rhode Island, now the third oldest program in the country. He remained actively involved in Hospice over the years, eventually serving as president of the board. He was the individual most responsible for lending credibility in the medical community to what was perceived in its early days as a counter culture movement of alternative medicine. He brought Hospice into the medical mainstream, where it remains today.

He was certainly the person who encouraged me to consider taking a less traditional path, both in medical school and then in my career as a physician. I am grateful for his guidance and his example of dedication.

August Zabbo, MD '80

I was always in awe of Dean Aronson's intellectual curiosity that extended beyond the usual medical sphere. By his own example he encouraged medical students to develop their pursuits and talents beyond medicine alone.

He approached each problem with genuine intellectual curiosity, rational analysis and wisdom resulting in clear unambiguous decisions.

* * *

After his ten-year stint as Dean, Dr. Aronson continued to demonstrate his timeless devotion to those in need of his professional and personal concern in so many significant ways - with the Hospice movement, lecturing to Brown students, serving on the State Licensing Board, writing weekly pieces for the *Providence Journal*, developing the unique Interfaith Health Care Ministries, serving as the editor of the *Medicine & Health/ Rhode Island*, and, most of all, offering his energy, love and humanity to friends, colleagues, present and former students, the community-at-large – literally all of us who are the better for having known him.

Ruth B. Sauber is Medical Student Affairs Officer Emeritus, Brown University School of Medicine.

Gratitude for a Long Personal and Professional Relationship

Edward Feller, MD

I am elated that at the age of 53 with much of my medical career stretching behind rather than ahead of me it remains possible to have a mentor, role model and friend as special as Stan Aronson. My contribution to Rhode Island medicine and local medical literacy likely consists of persuading Stan, while he was in a Miriam Hospital recovery room under the continued effect of sedation after minor surgery, to assume the role of Editor of the struggling *Rhode Island Medical Journal.* As Chairman of the Editorial Board, I have been privileged to work closely with this remarkable individual for 10 years.

Stan and I collaborated on an investigation of a 1763-64 epidemic on Nantucket that decimated the Native American population of the island. I was delegated to present our research at a national meeting. After Stan, Betty Aronson and I had dinner the night prior to our presentation, Stan remarked, “Let’s get a slide projector and go over your talk.” We spent two hours (two very late evening hours) dissecting each slide and every needed inflection until he was satisfied that this, perhaps my thousandth prepared talk, met his standards. A mark of a great and caring teacher is the ability to raise others to a level previously unachievable. Our presentation was a reflection of his unstinting devotion to excellence and his commitment to me that he cared about my success.

I have leaned on Stan for support and guidance throughout our long personal and professional friendship. This rumination represents my sole contribution to this journal that has not benefited from his stringent unerring editorial input. I have often sent him extra-sharpened red pencils lest he not have sufficient supplies to render my prose acceptable. Never has his tinkering failed to produce an improved result. I am proud and grateful that each issue bears the stamp of such an erudite, concerned expert mind.

Stan and I speak several times per week by phone and meet regularly to discuss the Journal. Our conversations tend to be wide-ranging, touching on a myriad of other subjects. I am continuously excited by his unflagging enthusiasm for medicine, his mastery of the written and spoken word, and his appreciation of the power and beauty of knowledge. He has also helped me as a staunch supporter during a prolonged and agonizing family illness. I am privileged to know his wonderful wife and count his daughter Lisa as a close valued friend. I am thankful that his life has touched and enriched mine.

Edward M. Feller, MD, is Chair of the Editorial Board, Medicine & Health/Rhode Island.

For Stan Aronson: My Once and Former Neighbor

Steven Davis ’78, MD ’82

He was a good neighbor
In a neighborhood of good neighbors —
Yankees, mostly, (in the good sense):
Quiet and solid and thoughtful.
You would not even have to ask them for what you
needed,
They already know and had it for you.

Even though he had been a city boy,
Erudite then
Erudite still,
He fit in:
The new kid on the block in a three hundred and
fifty-year-old town,
He took his turn humbly making the dump run on
Saturday morning
Unless he was off to Botswana or Oklahoma City to
assess a medical school,
And even then he would make it up when home.

He had a passion and a gift for horticulture
But also knew from first hand experience
That success in the garden and yard,
As elsewhere,
Is akin to Edison’s definition of genius:
1% inspiration and the rest hard work.
And for some reason (probably the final effect)
He and Betty liked to move rocks - big rocks - from
here to there and there to here,
(Not my idea of a good time)
To get just the right contemplative angle
Or shadow
Or eclipse.
You ought to walk through their arboretum
sometime.
Note the sculpture, note the craft, note the subtlety.
It will make you think
And ponder:
Man helping Nature to improve upon itself.

And even though our definition of a meadow
differed,
There were many things that we agreed on
Without talking about it:
How one treats a neighbor,
How one treats a colleague,
How one treats a patient, a person.

Ever the gentleman, ever gracious;
His kindness and expertise made the worst moment
of at least one person's life
Bearable.
He was there when it counted most.

If what your neighbors think
about you is your core,
Then his is gold, indeed.

Steven Davis, MD, is Clinical Associate Professor, Department of Family Medicine, (Memorial Hospital) Brown University School of Medicine.

A Doctor and a Preacher

Charles A. Baldwin

Aronson is one of those characters (and he is a character!) who enjoys not only conjuring up ideas, but also translating them into plans and programs. From my experiences, I offer three such instances:

1) Interfaith Health Care Ministries

When the Brown Program in Medicine was becoming a full-fledged Medical School affiliated with several hospitals which would become teaching and research institutions, Aronson was called to be the first Dean. I recall my first meeting with him in a discussion about religion and medicine (I thought he was an arrogant ignoramus), and ever since we have been fast friends. Out of that discussion and many, many others involving the several "helping professions" grew Interfaith, a program designed to introduce medicine and religion to each other, and more specifically, train clergy in the arts of pastoral care for the sick and dying. The program endures and thrives to this day. Stan has helped see to it.

2) Hospice Care of Rhode Island

Initially a movement begun in Great Britain to care for the terminally ill, Hospice sought to change societal attitudes toward death and dying, training up health care professionals and volunteers to provide the hands-on care and to establish the program and institutions which would pay ongoing attention to the special needs of this special population.

At a critical junction Aronson became the chairman of the Board of Hospice Care of Rhode Island, and he continues to this day to be a creative trouble-maker as a trustee.

3) Brown and Tougaloo

In 1963, Brown entered into a relationship with Tougaloo College, a small historically black college north of Jackson, Mississippi. (Key local players were Barnaby Keaney, Irving Jay Fain, and Larry Durgin). As this relationship grew with exchanges of faculty, students, administrators and trustees, Aronson saw an opportunity for the Medical School. He wanted to attract more minority students to Brown, and here was a black college with an unusual record in sending its students to graduate and professional schools. So, after much consultation between Brown and Tougaloo faculties, the Early Identification Program was born and lives to this day. The two faculties would identify two Tougaloo sophomores bent upon careers in medicine and, in effect, guarantee them admission to the Brown Medical School upon completion of the Tougaloo degrees.

This program has been a major factor in Brown's successful effort to attract African American students and has become a model followed by many other institutions in the United States.

Ideas and arguments, works and music, may stand on their own feet. Aronson appreciates them all.

He also thrives on translating ideas into policy, strategy and tactics, and this makes him unusual.

Charles A. Baldwin is Chaplain Emeritus, Brown University.

The Editor's Viewpoint

Robert Whitcomb

In 1992, my eagle-eyed wife, who was working with our friend Fraser Lang on some chores involving *Rhode Island Medicine*, the predecessor name of today's *Medicine & Health/Rhode Island*, brought to my attention the graceful work of Dr. Stanley Aronson, the publication's distinguished editor. Since that time, it has been my pleasure to develop a professional and personal friendship with this great physician and humanist - a link that has been one of the offsetting joys of a job that presents its fair share of the unfriendly.

After reading a few Aronson columns in *Rhode Island Medicine*, I was hooked. Here was the sort of Renaissance man who could write about virtually anything, and make it interesting, mordant, charming, hilarious, inspiring and profound by turns. Here was the physician as humanist. And he doesn't write about Washington scandals, and generally eschews public policy pronouncements. How refreshing!

His columns represent humanistic learning and eloquent writing for their own sakes, and are virtually never out of date.

No wonder Stan has developed a national name as a columnist. Indeed, after careers that have seen him become famed as a neuropathologist, as an expert in Third World epidemiology and as the founding dean of the Brown Medical School, as well as a founder of Hospice Care of Rhode Island and the Interfaith Health Care Ministries, he is now well into another career - national media star! Indeed, it is rather shocking to see this previously respectable member of one of the traditional professions ensnared by the unsavory show biz world of journalism.

What's he like to work with? Well, ribald, witty, courtly, avuncular, grandfatherly, brotherly, vaudevillian are some of the words that come to mind. But perhaps scholarly, authoritative and charismatic stick in the mind the longest. Indeed, so authoritative and charismatic is Stan that it didn't take long for the folks manning security in the lobby of the *Journal* Building to decide it was best to usher him directly to the elevator leading to my office. You don't harass a major religious figure! No security badge needed.

I have learned much about many things from Stan, especially from his weekly columns, but also from our all-too-brief early morning chats in my office and our all-too-rare meals out. I and our readers have learned things about history, and the development of medicine and langugage, that are just not available in the mass media these days - except, I am proud to say, in our little corner of it. (Stan even taught me a few things about a not-very-distant ancestor of mine, the physician James Young Simpson.)

I also have learned from Stan some extraordinary life histories that I probably could not have learned from anyone else. I learned how certain people were - and are - coping with afflictions of various kinds, and how they seek and find meaning amidst their daily suffering. And I learned something from Stan Aronson's compassion, grounded in realism and accepting of human foibles, and informed by a deep curiosity about his fellow man.

It is marvelous that Stan's writing put his wisdom, learning and elegant style at the disposal of millions of readers. It is, however, sad that few of them will ever have the opportunity to personally know this brilliant, kindly and resilient man. I am blessed to count myself among the lucky few.

Robert Whicomb is Editorial Page Editor, The Providence Journal, *and editor of* Health Care Horizon.

The Chairman of the Board's Viewpoint

Patricia Nolan, MD, MPH

Some people enhance the quality of every task they undertake. Dr. Stanley Aronson is such a person. A superb medical scientist and historian, Dr. Aronson always adds to our store of knowledge. He demands the best of those in medical practice, yet he brings personal warmth and humor to the difficult task of judging physician practice.

Dr. Aronson has been a part of the Board of Medical Licensure and Discipline for a full two terms. He has served on licensing committees, on investigating committees, and on hearing panels for the Board. He has been the vice-chairperson of the Board, providing continuity as Directors of Health change. He has sifted paper mountains of data to weigh the evidence and decide when a physician's practice has been unprofessional. In his willing participation in the work of the Board, he lends to our work his wisdom and understanding. As a result, the Board is effective in protecting the quality of care received by patients in Rhode Island and in assuring that the professional conduct of our physicians meets the standards of the profession.

Dr. Aronson does all this with a gentle manner, a smile, and a warm good humor that improves our judgment and maintains our balance.

Some people enhance the quality of every task they undertake. Dr. Stanley Aronson is such a person.

Patricia Nolan, MD, MPH, is Director, Rhode Island Department of Health, and Chairman, Rhode Island Board of Medical Licensure and Discipline.

The Objective Statistician's Viewpoint

Jay S. Buechner, PhD

Dr. Stanley Aronson served as Editor-in-Chief of *Medicine & Health / Rhode Island,* formerly *Rhode Island Medicine,* and prior to that *Rhode Island Medical Journal,* from July 1989 through December 1998. His tenure extended over a period of nine years and six months. In lieu of the monthly Health by Numbers submission, this report presents selected measures of the contents of the journal and of the participation in the journal by the state's health care community under his leadership.

Methods

The number of items published in the journal and the number of authors submitting items were abstracted from the annual author, subject, and title indexes for each volume of the journal, published in either the last issue of the volume or the first issue of the succeeding volume. For volume 81 (1998), a draft copy of the indexes was provided by the Managing Editor of the journal. Included as published items were invited and contributed articles, editorials, contributions to regular columns (Health by Numbers; Philately and Medicine; Vital Statistics; Medical Ethics, Law, and Health Policy; Judicial Diagnosis; etc.), features (Rhode Island Medical Journal Heritage, Necrology, etc.), reviews of books and videos, and letters to the editor, insofar as they were included in the indexes. Adjustments were made to account for changes over the years in the methods used to organize the annual indexes.

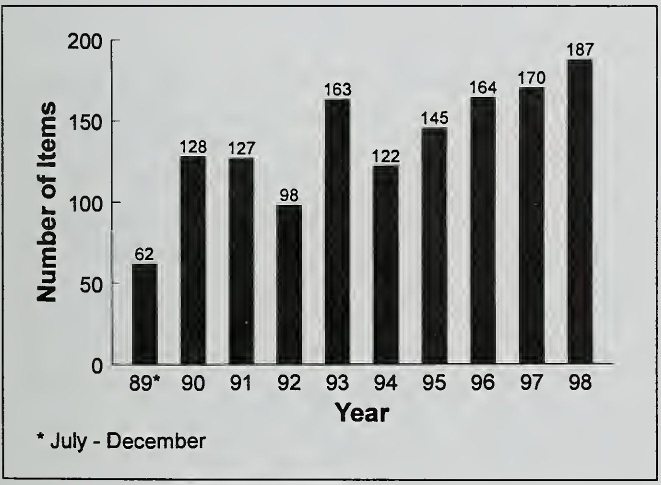


Figure 1. Number of Items Published in *Medicine & Health/Rhode Island,* by Year, 1989 - 1998.

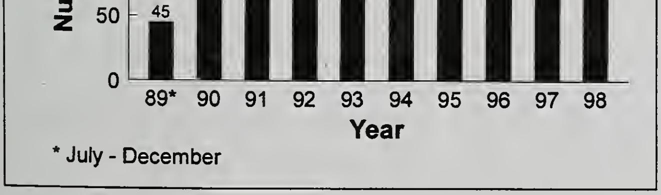


Figure 2. Number of Authors Contributing to *Medicine & Health/ Rhode Island,* by Year, 1989 - 1998.

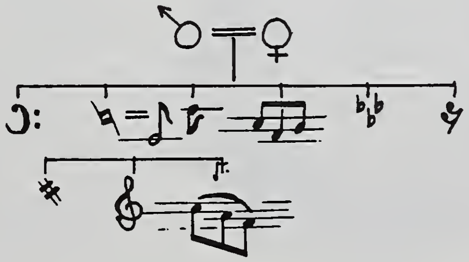
Results

During Dr. Aronson's tenure as Editor-in-Chief, a total of 1,366 listed items were published in the journal, or an average of 12.0 per month. The number of items published in each year ranged from a low of 98 in 1992 (excluding 1989, during which Dr. Aronson served for six months) to a high of 187 during 1998. (Figure 1) Similarly, the total annual number of listed authors ranged from 127 in 1990 and 1992 to a high of 223 in 1998. (Figure 2)

Discussion

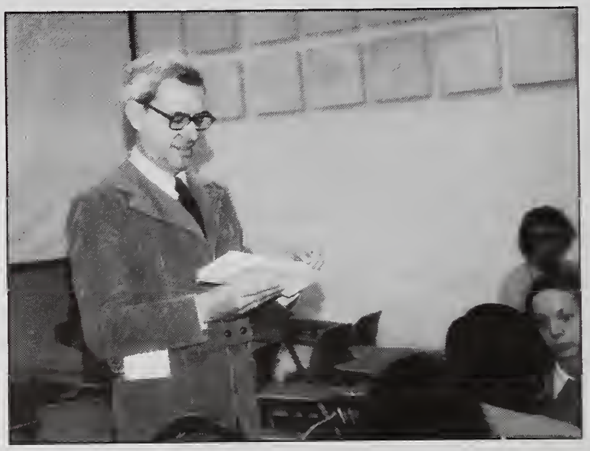
Dr. Aronson's editorship has been marked by growth in both the number and scope of the items published in the journal now entitled *Medicine & Health/ Rhode Island.* One objective he has adhered to consistently is the inclusion of public health issues in the journal's regular fare. Consequently, there are now monthly or other periodic columns that present issues in vital and health statistics, public health practice, and health policy. He has also added columns covering medical ethics, legal issues in medicine, and even philately and medicine. In the course of his service, he has broadened both the subject matter of the journal and the participation in authorship in the journal. He is leaving us a different and better publication than he found.

Jay S. Buechner, PhD, is Chief, Office of Health Statistics, Rhode Island Department of Health, and Clinical Assistant Professor of Community Health, Brown University School of Medicine.



A Challenge to Freud

Joan Retsinas, PhD



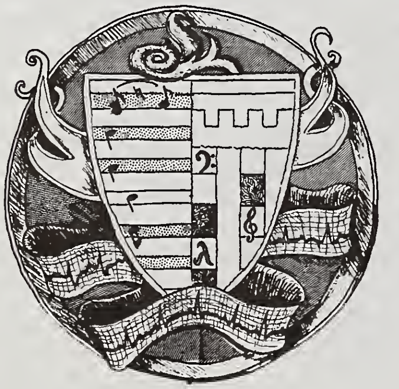
Sigmund Freud should have met Stanley Aronson. Freud, who posited the distinction - even a Maginot Line - between arbeit and lieben should have met a person who erased the line, who loves what he does, and embraces it, and the people around him, as a calling. Or more precisely (and Dr. Aronson would insist on precision) as part of his being, a sort of Heidegger-like Dasein, to meld metaphors (something Dr. Aronson would deplore).

I am thrilled to have met Stanley Aronson in the era beyond titles: he was no longer dean, neuropathologist, professor, but the editor of a tenacious, century-old Journal, seeking an administrative factotum to help with the minutiae. One time, exasperated with double-checking the titles, degrees, and affiliations of all the Journal contributors, I cited Emily Dickinson's lines about “I am Nobody, Who are You? Are You Nobody Too?” He understood, at least partly because he had once moved so stupendously successfully in the world of credentials, and could look on it with an ironic detachment. When I met him, the credentials had sloughed off, much like an outer covering, and the dizzying mixture of personality, character, wisdom, and humor emerged that was him.

And what a person he is!

His catholic range of interests has enriched not just *Medicine & Health/Rhode Island* readers, but thousands of Rhode Islanders through the publication of his historical essays in the *Providence Journal.* Whenever I mention that I work for the Medical Journal, people praise his columns - mentioning the one about Danbury Hatter's Disease, or the Pontine Swamp, or the Mayo brothers. Medicine is not the sole purview of physicians; and Dr. Aronson's clear eloquent prose makes the sometimes accidental progress of medicine real to all kinds of readers.

His enjoyment of that history also comes through to readers. Medicine's halting progress toward better health is inherently interesting, inherently worthy of attention and respect. In the historical retrospective page of the *Journal* (“Ninety Years Ago,’ “Fifty Years Ago,” Twenty-Five Years Ago”) he recounts, ever so gently, the key findings and conclusions that from today's perspective seem bizarre.



Readers too can glimpse the wonderful sense of humor that pervades his perspective. In the ironic phrase tucked into a paragraph, they can sense the smile on his face even as he types away.

Certainly that sense of humor has suffused our relationship, as editor and factotum. Each month the Journal emerges from a tangle of mistakes - mine, the printer's, the mailer's, the advertiser's. And that is after Dr. Aronson has edited the contributions themselves for mistakes. Although individual mistakes are of course avoidable, their presence - some months more than others - seems part of the beast, testimony to human foibles. Some months we are awash in foibles. Dr. Aronson never gets angry at me or anybody else in the production hierarchy, never blames anybody, never is unduly unruffled. As a historian, he understands the foibles of historical people; surprisingly - and refreshingly - he understands flesh and blood people too. He looks upon the monthly mishaps with the kindly good humor that springs from genuine appreciation of the human condition.

I confess that I don't love most of the administrative tasks of being managing editor. When I call up a contributor to ask about reference 8 (“What is the volume number?) or ponder the production schedule with the printer (“Can we move this up a day?”), I am doing a job. Most of the time I have my own Maginot Line separating arbeit from lieben. Yet I have grown inordinately fond of Stanley Aronson; and sometimes, when I'm talking to him about a cover, or going over revisions of second-stage galleys, or pondering an obscure word - I too love this job. For a moment I, like him, bridge arbeit and lieben.

Joan Retsinas, PhD, is managing editor, Medicine & Health/Rhode Island.

Medical Complications in Patients with Traumatic Brain Injury

Jon Mukand, MD, PhD, and Bernadette Santos, MD

Abbreviations Used:

ADH	antidiuretic hormone
EHDP	etidronate disodium
HO	heterotopic ossification
LDUH	low dose unfractionated heparin
NSAID	nonsteroidal anti-inflammatory drug
PTH	post-traumatic hydrocephalus
PE	pulmonary embolism
PTS	post-traumatic seizures
ROM	range of motion
TBI	traumatic brain injury

Medical complications in patients with traumatic brain injury (TBI) are varied and difficult to assess because of cognitive and communication deficits. Prompt identification and treatment of these complications can shorten the inpatient stay, minimize disability,[1] and improve patients' quality of life. This article discusses some of the most common medical issues in patients with TBI, including neurologic, cardiopulmonary, gastrointestinal, urologic, vascular, musculoskeletal, and endocrine. Cognition, sexuality, pain and spasticity are important problems, but are beyond the scope of this article.

NEUROLOGIC

Post-traumatic seizures

Post-traumatic seizures (PTS) occur in 5 - 7% [2] of hospitalized patients with TBI and may be classified as early (during the first week) and late (after the first week). Patients with acute intracranial hemorrhage and depressed skull fractures are at increased risk for developing late PTS.[3] Other risk factors include dural tears, focal neurologic signs, post-traumatic amnesia greater than 24 hours, and the presence of a foreign body.[2,3,4]

A large study published in 1990 found no benefit in preventing late PTS by using phenytoin beyond the first week.[4] Most TBI specialists no longer use seizure prophylaxis beyond the first two weeks unless there is a documented seizure. The American Academy of Physical Medicine & Rehabilitation recently endorsed the following practice guidelines for the use of anticonvulsants in this population. Phenytoin is recommended for prophylaxis only in the first week post-injury in those with identified risk factors. Carbamazepine is the drug of choice in tonic clonic and partial complex seizures. Valproic acid, phenytoin, and phenobarbital are effective in tonic clonic seizures, but the latter has significant adverse effects on cognition.[5]

The cognitive impairment resulting from anti-epileptic drugs is of great concern in these patients. There is minimal difference in cognitive side effects from phenytoin, carbamazepine, and valproic acid.[6] Newer antiepileptic drugs await clinical evaluation in the TBI population.[5]

Post-traumatic hydrocephalus (PTH)

This complication has been estimated to occur in 1– 8% of patients.[7] A newer study found an incidence of 20% in patients with severe TBI and 45% in those with ventriculomegaly.[6] Impairments in flow and absorption of cerebrospinal fluid lead to the development of PTH, especially in patients with subarachnoid hemorrhage.[1] Symptoms range from deep coma to the triad of normal pressure hydrocephalus (ataxia, dementia, and urinary incontinence). Typical presentations include prolonged coma or impaired functional recovery. PTH may also present with spasticity, emotional lability, and seizures. Monthly CT scans are still the most reliable for diagnosing progression of ventricular dilatation.

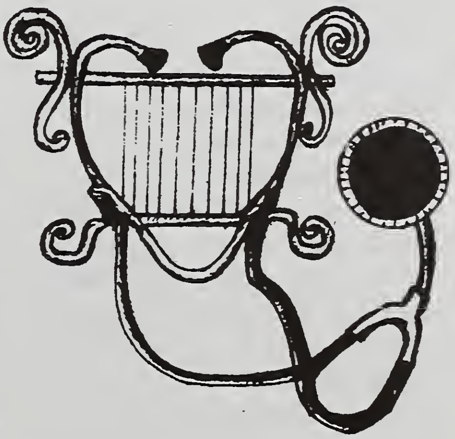
Benefits from neurosurgical intervention depend upon diagnostic and clinical criteria. Shunting is found to be successful in patients with CSF pressure greater than 180 mm Hg.[7] Psychometric measures and gait testing before and after a lumbar puncture may be useful. Improvement in two out of four tests (gait, reaction time, perception, and memory) following removal of 50 cc of CSF is a good prognostic sign for shunting.[1]

CARDIOPULMONARY

In severely head-injured patients, EKG changes are common and frequently indicate diffuse myocardial ischemia.[1] Arrhythmias may also occur because of a catecholamine surge or directly related to the cerebral damage. CPK-MB and serial EKGs are used to assess myocardial involvement.

Respiratory complications are common because of the need for mechanical ventilation, especially in severe injuries. Most patients require nasal or oral intubation followed by tracheostomy, resulting in bacterial colonization as well as pneumonia. Concomitant neurologic deficits also compromise pulmonary toilette, and severely impaired patients need a long-term tracheostomy because of the high mortality rate associated with decannulation.[8]

Laryngeal and tracheal complications include vocal cord paralysis, tracheal



stenosis, sub-glottic and glottic stenosis, and tracheomalacia. Tracheal and subglottic stenosis may occur weeks after extubation, typically after an upper respiratory infection, and are characterized by respiratory distress and biphasic stridor.[8]

GASTROINTESTINAL

Elevated liver enzymes are the most common GI disorder in the TBI population, occurring in up to 43%.[3,8] Direct trauma to the liver, infection, drugs, alcohol use, and blood transfusion are some of the etiologies.

These patients have high nutritional stress and frequently have a major weight loss.[8] Adequate nutrition is initially supported by parenteral or enteral means and some patients require a long-term percutaneous endoscopic gastrostomy or jejunostomy tube.

Dysphagia occurs in up to 27% of TBI patients and a study has shown that 81% had delay or absence of the swallowing reflex, 50% had diminished tongue control, and 33% had reduced peristalsis. Patients frequently have reduced arousal and cognitive problems. Once they are more alert, a swallowing evaluation, and a modified barium swallow can be conducted. Anorexia and hypophagia are not uncommon, and megestrol may be used with monitoring of its side-effects, including nausea, thrombo-embolic phenomena and pituitary/adrenal axis abnormality.[9]

Delayed gastric emptying can also contribute to poor oral intake. Among its causes are gastric outlet obstruction, anticholinergic medications, sympathetic or parasympathetic dysfunction, endocrine abnormalities, and immobility. The following is a review of some of the medications used to improve gastric motility.

Metoclopromide is a dopamine receptor antagonist and acts as an upper GI stimulant. Side effects include confusion, sedation and extrapyramidal syndromes, which make it less suitable for the TBI population. Cisapride increases acetylcholine release into the gut, accelerating gastric emptying, and intestinal transit. It is structurally related to metoclopromide but has fewer extrapyramidal side effects.[9]

Ketorolac is a nonsteroidal anti-inflammatory drug (NSAID) that inhibits the cyclooxygenase pathway to limit prostaglandin synthesis. Some studies on patients who developed post-operative ileus were found to have high levels of prostaglandin. Following administration of ketorolac, ileus resolved. NSAIDs also depress C fiber activity, inhibiting transmission of nociceptive impulses through the spinal cord and shortening post-op ileus. Adverse effects include GI, renal and hematologic complications but these can be minimized if drug use is limited to 14 days.[9]

The combination of erythromycin and octreotide has been shown to improve gastric emptying. Erythromycin controls the bacterial overgrowth that can worsen gastric stasis due to nausea, vomiting and bloating. At the same time, it releases motilin, a neuroactive peptide that enhances GI motility.[9]

Patients with TBI are at high risk for gastrointestinal bleeding due to stress ulceration.[7] Cigarette use, alcohol, *H. pylori* and psychologic factors may increase secretion of gastric acid and pepsin. Mucosal defensive factors include secretion of a glycoprotein mucosal gel that stimulates secretion of bicarbonate, maintains mucosal blood flow, and renews epithelial cells in response to mucosal injury.[9] Drug therapies include antacids and adsorbents, H2 blockers, acid secretion inhibitors, and acid pump inhibitors.

Aluminum and magnesium antacids increase the pH of gastric secretions, thereby decreasing the effects of acid on vulnerable gastric mucosa.[9] H2 blockers inhibit the action of histamine on H2 receptors of parietal cells to reduce gastric acid output. Cimetidine, famotidine and nizatidine have been shown to be effective, with famotidine having the least CNS side effects. Ranitidine, though effective, has a lot of side effects including drowsiness, confusion, headache, fever, diarrhea, rash, dysrhythmias, and elevation in LFTs.[9]

Misoprostol is a synthetic prostaglandin E1 analog that inhibits acid secretion. It is frequently used for prevention of NSAID-induced ulcer. Omeprazole binds to the acid pump in gastric parietal cells to inactivate the final step in HCl secretion. It has been shown to be the most effective drug for peptic ulcer disease.[9]

UROLOGIC

Urinary incontinence is a common problem in patients with TBI, and can have a profound impact on the recovery process. Urinary tract infection should be ruled out because antibiotic treatment may lead to continence. Urinary incontinence may result from bladder or bladder outlet dysfunction.[9]

Hyperactivity of the detrusor muscle often improves with anticholinergic medications such as oxybutynin, propantheline, and tricyclic antidepressants. These have been shown to suppress uninhibited bladder contractions, increase bladder capacity, and increase urethral resistance.[9]

Urinary retention may result if the bladder is allowed to become distended beyond its normal capacity of 500 ml. Bethanecol directly stimulates the muscarinic receptors and may restore detrusor tone and function. If retention is due to a bladder outlet or sphincter problem, alpha-adrenergic blocking agents such as phenoxybenzamine, prazosin, terazosin, and uropidil may be effective. Patients should be monitored for orthostasis because many are already at risk of falls.

Alpha-adrenergic agonists such as ephedrine and phenylpropanolamine may control incontinence by stimulating the internal sphincter. Caution must be used in patients with detrusor hyperreflexia or poor bladder compliance because of the possible development of reflux and hydronephrosis.[9]

VASCULAR

Venous thromboembolic disease accounts for significant morbidity and mortality with an estimated incidence rate of 20–54%.[9] Risk factors for the development of DVT include fractures of the pelvis, spine and lower extremities; coma and prolonged immobility; femoral vein catheterization; sepsis, shock, and transfusion. Relative risk factors include previous DVT, coagulopathy, varicose veins, obesity, CHF, MI, arrythmias, cancer, pregnancy, and oral contraceptive use.

Signs and symptoms are highly unreliable, with only 1.5% having clinical evidence of the disease.[10] Pulmonary embolism resulting in sudden death is the finding in 70-80% of TBI patients with thromboembolic disease,[9] so early detection and prophylaxis is critical. Prophylactic treatments may be classified as mechanical and chemical. Mechanical devices include compression elastic stockings, intermittent pneumatic compression, and filter placement in the inferior vena cava. The use of elastic stockings and intermittent pneumatic compression is limited, especially in patients with lower extremity fractures, external fixator devices and major wounds.[9]

Chemical agents include low dose unfractionated heparin (LDUH), low molecular weight heparin (LMWH), warfarin, and aspirin. The efficacy of LDUH has been established in general surgery, neurosurgery and some trauma populations but not in the TBI population.[9] The ideal dosing and timing are unknown, and the major concern is the risk of bleeding. Early studies suggest less bleeding with LMWH than LDUH but equal thrombolytic effect. Clinical studies in general surgery, orthopedics, stroke, and trauma populations report bleeding rates from 2 – 12%.[9]

Warfarin has limited use in the TBI population because of the delayed pharmacologic effect, need for close monitoring, and high risk of hemorrhage. Though aspirin has some benefit in preventing DVT, it has not been investigated in the TBI patient.

Treatment options for DVT above the calf and pulmonary embolism (PE) include unfractionated heparin or LMWH followed by warfarin and IVC filter placement. These options are not always effective, and occasionally PE occurs despite treatment. For instance, unfractionated heparin followed by warfarin prevents fatal PE in 95% of patients.[9] There is currently insufficient data regarding the extent and duration of the bleeding risk associated with anticoagulation following TBI. An unacceptable risk of intracranial hemorrhage prohibits full anticoagulation in the first 7 to 14 days post-injury.

Absolute contraindications to full anticoagulation include unsecured aneurysm following subarachnoid hemorrhage, hematomyelia, active GI bleeding, and intracerebral bleed.[9] Relative contraindications include severe hypertension, retinopathy, and hemophilia. IVC filter placement is the best alternative when anticoagulation is unsafe. Complications from IVC filter include chronic edema, postphlebitic syndrome, filter occlusion, clot propagation, and filter migration and fracture. Some patients may benefit from custom-fitted compression stockings or low-dose warfarin after the acute phase.

The TBI population is complex and has a variety of unusual medical complications.

To prevent recurrence, warfarin treatment is continued for 6 months for PE, 3 months for proximal DVT and 1.5 to 3 months for calf thrombus. Warfarin should be discontinued when feasible because there is a positive correlation between duration of therapy and hemorrhagic complications.

HETEROTOPIC OSSIFICATION

Ectopic bone forms around major joints and, when untreated, can delay and prolong rehabilitation. The incidence of heterotopic ossification (HO) ranges from 11 – 76% depending on the study population and method of detection. Risk factors include coma of more than 2 weeks, spasticity, and long bone fractures. The physical findings include pain, decreased range of motion, swelling, increased local temperature, systemic fever, erythema, and increased spasticity. Complications from HO include joint contracture and ankylosis,[3] vascular compression, and lymphedema. The greatest risk for developing HO is during the first 2 – 6 months after injury in patients with increased tone and comatose for at least 3 weeks.[9] Prophylactic measures include range of motion (ROM) activities, spasticity management, etidronate disodium (EHDP), and NSAIDs.

Etidronate inhibits the precipitation of calcium phosphate, transformation of calcium phosphate into crystalline hydroxyapatite, the aggregation of hydroxyapatite crystals, and crystal growth. It does not affect the bone matrix formation that is the initial phase of HO development.

Etidronate appears to decrease the incidence and severity of HO following spinal cord injury (SCI) and TBI. Its long-term ability to prevent HO in patients with TBI is yet to be established. Long-term studies in patients with SCI indicate that HO may increase after cessation of etidronate but to a lesser degree than in the control group.[9]

Gastro-intestinal complications from EHDP include nausea, vomiting, and diarrhea, but these are resolved by dividing the daily dose. Osteomalacia and long bone fracture may occur with use of greater than 6 months.

The mechanism by which NSAIDs suppress HO development is unknown. Following trauma, local remodeling takes place through an inflammatory reaction and induces ectopic bone growth. NSAIDs influence this process by inhibiting prostaglandin synthesis. Medications studied include ASA, diclofenac, salicylate, and ibuprofen. The duration of treatment is uncertain but may range from 6 weeks to 6 months depending on continued risk for HO. Potential contraindications include peptic ulcer disease, renal impairment, and hepatic dysfunction.

Once HO is established, pharmacologic treatment is not effective. Treatment options include joint manipulation under anesthesia and surgical resection in combination with radiation and NSAIDs.

ENDOCRINE

Endocrine complications occur in 25% of patients with moderate to severe head injury. Neuroendocrine disorders are related to the hypothalamic pituitary axis, autonomic regulation, and appetite homeostasis.

Vasopressin or antidiuretic hormone (ADH) is released from the posterior pituitary in response to low blood volume. It then acts on the distal tubule and collecting duct of the kidney to increase water resorption. After TBI, excessive ADH secretion can cause SIADH. Patients frequently present with hyponatremia, and a high urine osmolality compared to the serum osmolality. Patients become symptomatic when the serum sodium goes below 125 meq/L or if it drops rapidly; elderly or very young patients become symptomatic at 130 meq/L. Nausea, malaise, lethargy, and cramps may progress to psychosis, seizure, and coma when hyponatremia is not addressed.

At sodium levels of 116-124, water restriction of 500 to 1000 ml per day is instituted. Demeclocycline may be added if water restriction is ineffective but caution should be exercised in patients with hepatic dysfunction. Furosemide and salt are given for levels less than 115 meq/L. Rapid correction should be avoided because of possible central pontine myelinolysis; the sodium should only rise 20 meq/L in the first 24 hours of therapy.

Central diabetes insipidus in TBI occurs less commonly than SIADH.[1] ADH deficiency usually occurs as a result of CNS injury or complication of neurosurgery. Patients have urine output up to 3 – 10 L/day, causing the serum to become hypertonic and hypernatremic. Subcutaneous or intranasal aqueous arginine vasopressin (DDAVP) increases urine concentration and improves fluid balance.

Hypopituitarism is rare and is usually associated with functional and cognitive decline. Its insidious development is unclear and a plausible explanation is the use of high dose steroids used for the management of severe trauma. Manifestations are numerous and are related to multiple target organs. The prospective evaluation of hypothalamic anterior pituitary dysfunction begins with screening tests like TSH, free thyroxine index, testosterone, LH, FSH, estradiol, fasting prolactin, and cortisol. Replacement of the target gland hormones should be provided as indicated by testing and symptoms.

Adrenal insufficiency may result from direct trauma to the pituitary gland, or compromised vascular supply due to increased intracranial pressure. This condition is usually seen in severe cases of head injury, with cortisol levels notably low two hours after injury.[11] Central hypoadrenalism is rare; derangement in the diurnal rhythm of cortisol is more common. Symptoms include lethargy, anorexia, nausea, vomiting, and abdominal pain. Treatment consists of oral prednisone.

Hypertension is often associated with temperature abnormalities, tachycardia and hyperhidrosis due to the release of central catecholamines.[11] Intrinsic renal disease, endocrine abnormalities, and pheochromocytoma should be ruled out. Other causes include direct damage to the autoregulating centers,[8] elevation in intracerebral pressure, changes in cerebral blood flow, and medications. Treatment involves addressing the cause of hypertension. Beta-blockers have been successful; atenolol is better than propanolol because it has fewer cognitive side effects owing to its hydrophilic nature. Peripheral vasodilators should be avoided because they increase intracranial pressure and lower the cardiac index.[11]

Temperature dysregulation can occur either as central fever or hypothermia. The anterior hypothalamus contains temperature-sensitive neurons and the posterior hypothalamus controls thermoregulation via the autonomic nervous system. The evaluation should exclude infectious etiologies and drug reactions. Antipyretics and physical measures are helpful for fever reduction while a beta-blocker is effective in controlling tachycardia resulting from catecholamine surges.[11]

The hyperthermic syndrome occurs in patients with head injury and hydrocephalus who present with fever, hypertension, sweating, and tachycardia. Treatment involves surgical correction of the hydrocephalus, bromocriptine, morphine and anticonvulsants.[11]

Anorexia and hypophagia have been discussed earlier. Hyperphagia may result from bilateral temporal lobe damage, causing bizarre disinhibition, and exploratory behavior. Treatment includes appetite suppressants, SSRIs, and even methylphenidate because of its anorexic side effects.[9]

CONCLUSION

The TBI population is complex and has a variety of unusual medical complications. These require close monitoring in a neurorehabilitation setting, where specialized staff provide intensive rehabilitation for the physical, cognitive, and behavioral disabilities associated with TBI.

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THE CREATIVE CLINICIAN: CASE OF THE MONTH

"The practice of medicine is an art, not a trade; a calling, not a business ..." – WILLIAM OSLER, Aequanimitas

Editor: Anthony Mega, MD; Co-editor: Fred J. Schiffman, MD

Eastern Equine Encephalitis: Case Report and Literature Review

Rowena Emilia J. Tabamo, MD, and John E. Donahue, MD

Abbreviations Used:

CT	computed tomography
CNS	central nervous system
CSF	cerebrospinal fluid
DEM	Department of Environmental Management
EEE	eastern equine encephalitis
EEG	electroencephalogram
ELISA	enzyme linked immunoassay
HSE	herpes simplex encephalitis
IFA	immunofluorescence antigen
IgM	immunoglobulin M
MRI	magnetic resonance imaging
PCR	polymerase chain reaction

During the summer and fall, Eastern Equine Encephalitis (EEE) poses a public health threat in the Northeast.[1,2] It is a mosquito-borne arboviral (arthropod-borne) infection caused by the EEE virus, which is a single stranded RNA virus of 11,700 bases. It belongs to the genus *Alphavirus* which includes Western Equine Encephalitis and Venezuelan Encephalitis . The different arboviruses known to cause encephalitis have a varied geographical, vertebrate reservoir host and insect vector distribution.[1,4,7] EEE has been detected in man, horses, wild birds and mosquitoes from Florida to Canada.[4]

We present a Rhode Island case that occurred at an unusually early time.

CASE REPORT

On June 17, 1998, a 44 year-old male presented with the acute onset of headache, malaise, fever (106 °F), chills, vomiting and disorientation. Except for his increasing confusion, he had no neurological abnormalities.

His initial cerebrospinal fluid (CSF) had 61 white blood cells with 76% lymphocytic predominance, a protein of 259 and a glucose of 86. The serum glucose was 155. Arboviral serology and CSF culture studies were negative. His serum electrolytes were within normal limits except for hypokalemia and hypophosphatemia. His brain CT scan showed an unremarkable non-contrast study on the first day.

On June 19, his brain MRI with gadolinium showed diffuse cortical enhancement with increased signal intensity extending along all gyri, consistent with meningoencephalitis. He then developed a rapidly progressive mental status decline, generalized seizures and hypotensive episodes. On June 20, he was comatose despite treatment with Acyclovir and Ceftriaxone. He died on June 21.

His initial post-mortem studies showed negative arboviral serology, and virus could not be isolated from the CSF. Diagnosis was made post-mortem by immunofluorescence (IFA) on brain tissue using an EEE-viral specific monoclonal antibody and by polymerase chain reaction (PCR) specific for EEE.

REVIEW OF LITERATURE

The earliest reported human cases of EEE occurred in southeastern Massachusetts in July 1938. The illness was first recognized in horses. Most of the horse deaths occurred in a 30-mile square area extending from the mouth of the Taunton River in the south, to Boston on the north, and from the boundary of Rhode Island eastward to the coast of Massachusetts. The rainfall there during that summer and early autumn was unusually heavy, with many mosquitoes. Around the same time, a deadly type of human encephalitis appeared in the vicinity, largely limited to children, with the reported deaths in horses occurring a few weeks prior to the onset of human cases.[5] Although the prevalence rate of disease was higher in horses than humans, the rise and fall of the outbreaks in both groups appeared similar.[6]

Epidemiology and Incidence:

EEE has an incidence of 1% of the total viral encephalitides in the US and 2-3% among the arboviruses. Its current mortality rate is approximately 30-50% compared to an estimated 70% case fatality rate in 1938.[1,2,7]

Mode of Transmission:

The EEE virus is perpetuated in a bird-mosquito cycle. Its incidence tends to be higher in summers following several years of abnormally heavy rainfall, as wet conditions are believed to favor expansion of the mosquito population.[2]

In the North Americas, *Culiseta melanura* is the principal vector of EEE. It is a species of mosquitoes that feeds exclusively on birds. Avian EEE infection results in either fatality or a subclinical infection that results in viremia of sufficient magnitude to maintain a reservoir.

The virus is passed from infected mosquitoes to mammalian hosts by "bridge vectors." These are mosquitoes of broad feeding preferences - feeding on both birds and mammals. In Rhode Island, the EEE virus has been isolated from pools of several species of these bridge vectors, namely, *Aedes vexans*, *Aedes canadensis*, and *Coquilletedia perturbans*

This complex cycle explains why human EEE cases typically occur late in the season: it takes time for *Cs. Melanura* populations to increase and for the virus to build up in bird populations to the point where it might be picked up by bridge vectors.[8]

A bite from an infected mosquito vector results in viral deposition in subcutaneous and cutaneous tissues.[9] The incubation period is 5 to 15 days. Initially, there is marked viremia and a febrile response, which signals the replication of the virus systemically. The central nervous system (CNS) is affected via hematogenous spread with pathological lesions occurring throughout the brain and spinal cord, particularly severe in the cortex and basal ganglia.

Clinical Manifestations:

Illness usually begins with generalized symptoms of malaise, fever, anorexia and vomiting. The illness is usually subclinical and lasts a few weeks with complete recovery if there is no clinical CNS involvement.[4] CNS involvement begins with a slightly altered level of consciousness and disorientation. These findings are followed quickly by the appearance of nuchal rigidity and deterioration in consciousness to the point of coma by day two in the clinical illness. Seizures and focal neurological findings may occur.[7]

In a study of 36 EEE cases from 1988 to 1994 Deresiewicz reported the following initial signs and symptoms: fever, headache, nausea, vomiting, with ensuing confusion, somnolence, focal weakness, occasional cranial nerve palsies, seizures and meningeal signs with subsequent rapid deterioration to stupor and coma in days. Thirteen patients died.[14]

Prognostic Features and Long-Term Outcome:

Among the alphavirus infections, EEE causes the highest mortality rate. In severe cases, death occurs within days.[4] Children under 10 years of age have a better survival rate, hence a greater chance of developing severe deficits-mental retardation, seizures, personality disturbances, blindness, deafness, speech disorders and hemiplegia[1]

A longer prodrome with nonspecific , non-CNS symptoms leads to a milder clinical course, while a shorter prodrome with rapid progression to the comatose state (four days or less after initial onset) leads to death or a bad neurological outcome. It is thought that a longer prodromal period allows the patient's immune system to build adequate antibodies against the virus.[7]

LABORATORY CRITERIA FOR DIAGNOSIS OF EEE

(1) Fourfold or greater increase in serum arboviral antibody titer, between the acute and convalescent phase (note: because of the rapid course of disease, sera should be obtained at 2-3 day intervals during the acute phase of illness), or

(2) Viral isolation or demonstration of viral antigen by immunofluoresence antibody studies (IFA) and enzyme linked immunoassay (ELISA), or genomic sequences (PCR) in tissue, blood, cerebrospinal fluid or other body fluid, or

(3) Specific immunoglobulin M (IgM) antibody in CSF by enzyme imunoassay (EIA) antibody in CSF or serum. Serum arboviral IgM antibodies alone should be confirmed by demonstration of IgG antibodies by another serologic assay (eg neutralization or hemagglutination inhibition or immunofluorescence)[10]

OTHER LABORATORY/SUPPORTIVE DATA

CSF Analysis

Przelomski described abnormal CSF findings in 16 EEE cases, which showed pleocytosis , often neutrophilic.[1] Initial CSF wbc ranged from 11 to 4,000, with an average of

Table 1: Differential Diagnosis

I. Viral Agents
    - Mumps virus
    - Lymphocytic choriomeningitis virus
    - Epstein-Barr Virus
    - Cytomegalovirus
    - Adenovirus
    - Rabies

II. Non-viral infections resembling arboviral encephalitides
    - Tuberculosis
    - Cryptococcus
    - Histoplasmosis
    - Coccidioioidomycosis
    - Rocky Mountain Spotted fever
    - Leptospirosis
    - Toxoplasmosis
    - Lyme
    - Malaria
    - Trichinosis
    - Naegleria
    - Mycoplasma pneumonia
    - Cat scratch disease

III. Neoplastic , granulomatous , inflammatory states
    - Behcet's disease
    - Reye's syndrome
    - Acute Multiple Sclerosis
    - Systemic Lupus Erythematosus
    - Post-infectious encephalomyelitis

910. There was neutrophilic predominance in 2/3 of cases. Out of the 16 patients, 7 had more than 500 CSF rbc, 3 had less than 500, 6 had none.

The protein was increased to greater than a 100 mg/dl units in 25% of cases; however, the majority were in the 45-100 range. The CSF/Serum glucose ratio was <0.6 in 9 out of 10 patients and <0.5 in 4 out of 10 patients. Viral isolation from the CSF culture was rarely successful.

EEG

This may be useful in differentiating EEE from herpes simplex encephalitis (HSE). In EEE, the electroencephalogram (EEG) may show nonspecific background slowing, whereas periodic complexes in the temporal frontal regions are more specific for HSE. The EEG is a predictor of poor outcome in either disorder if it shows severe patterns such as burst suppression or disorganized background with delta slowing.[1]

NEURORADIOLOGY

Neuroradiographic studies of 32 EEE cases were nonspecific and showed abnormalities in the basal ganglia, thalamus, brain stem and cortex. Periventricular focal lesions and meningeal abnormalities were less frequent.[9,14]

PATHOLOGICAL FINDINGS

The pathological lesion of EEE is meningoencephalitis exhibiting three features: (1) a vascular component consisting of perivascular cuffing and arteritis, (2) a glial response including varying degrees of necrosis and (3) neuronal degeneration and neuronophagia. These changes are not pathognomonic. EEE tends to be more severe and more destructive than other arboviral encephalitides with predilection for the basal ganglia.(2)

Occasionally, virion particles may be seen in the extracellular space or in cytoplasm of macrophages by electron microscopy.[4,11]

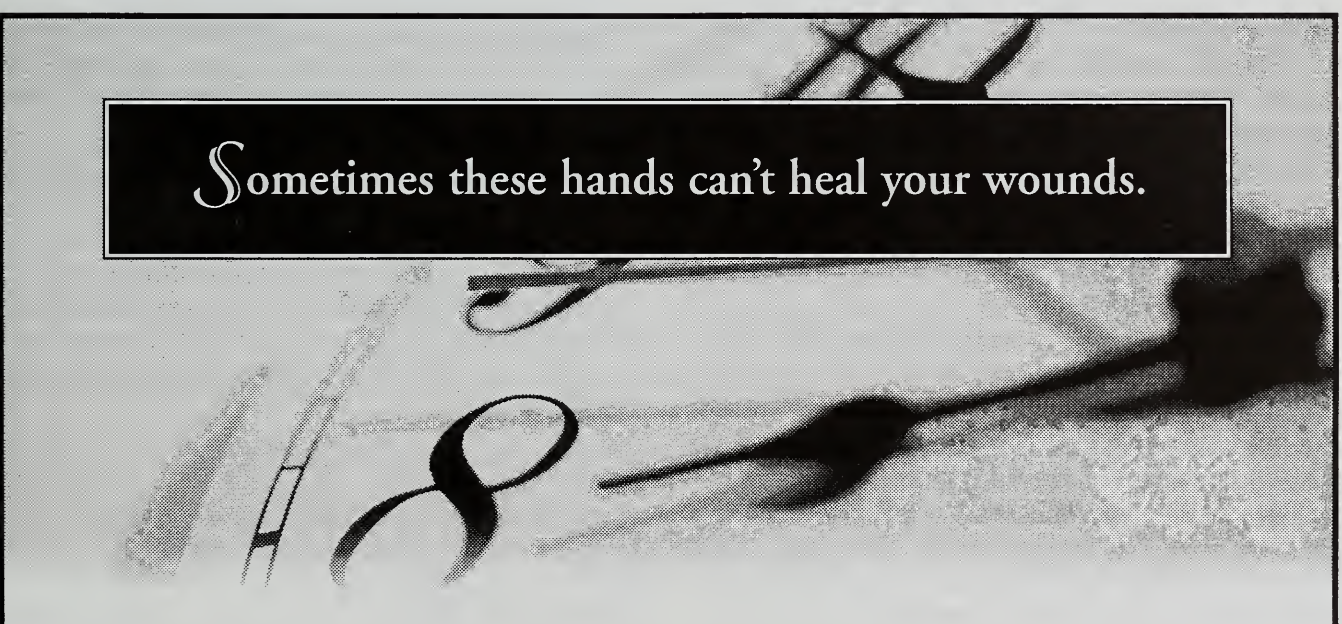
For this case, the microscopic studies showed diffuse hypoxic and ischemic changes and perivascular lymphocytic infiltration. No viral particles were visualized on electron microscopic studies.

Differential Diagnosis

Arboviral encephalitis may be clinically and pathologically indistinguishable from other forms of viral encephalitis. Focal brain involvement has been described in EEE. CSF studies may be non-specific.[3]

The differential diagnosis is large. (See Table 1)[3]

A history of mosquito exposure, the presence of an outbreak of similar disease in the community in the summer-fall seasons and a rapidly progressive clinical course are principal clinical clues to an EEE diagnosis but the diagnosis usually rests on serological and post-mortem studies.



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MANAGEMENT

There is no known effective antiviral therapy for EEE. Vigorous supportive therapy and avoidance of complications are crucial. Seizures should be treated with antiepileptic medications. Given the potential for the syndrome of inappropriate anti-diuretic hormone, serum electrolytes should be monitored periodically. Isolation precautions are not necessary.

Prevention depends on the control of mosquito vector populations and avoidance of mosquito exposure. Formalin-inactivated vaccines against EEE are available for limited human use for high risk laboratory workers but are of uncertain efficacy.[2,9] Veterinary vaccines are used against EEE in horses and birds. Appropriate use of insecticides may be effective in threatened or established outbreaks. Surveillance measures for breeding areas of mosquito vectors may serve as a basis of insecticide deployment. In the summer and fall, the Rhode Island Department of Environmental Management (DEM) and the Department of Health (DOH) monitor the presence of EEE weekly in different counties in Rhode Island, by trapping mosquitoes.

DISCUSSION

This was the first and only case of EEE reported in 1998, as of September. The last reported case in Rhode Island was in 1993. This case is particularly interesting in that it occurred earlier in the summer than expected. Despite the heavy rainfall this year, no virus has been isolated from mosquito populations, even in the area presumed to be the site for the patient's mosquito exposure.

Though a rare disease, the rapid clinical course resulting in death despite optimum supportive therapy continues to pose a health concern. No treatment is available and clinically diagnostic measures are usually of low yield. The diagnosis rests entirely on laboratory results because clinical findings are non-specific.

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Rhode Island
Quality Partners, Inc.

Myocardial Infarction

Lawrence Proano, MD, FACEP, and Liudvikas Jagminas, MD, FACEP

INTRODUCTION

In 1992, myocardial infarction was identified as the second leading cause of admissions to hospitals for Medicare beneficiaries in the United States. The average stay for this diagnosis cost $11,650 and represented 15% of all Medicare expenditures for short-stay hospitalizations.[1] In fiscal year 1996, Rhode Island Quality Partners' data for Medicare patients showed that myocardial infarction was seventh leading cause for admissions to Rhode Island hospitals.

There is significant scientific evidence that timely, appropriate interventions improve survival, improve the quality of life, reduce the need for interventional procedures (angioplasty, bypass grafts), and reduce the incidence of subsequent heart attacks.[2] For example, a recent survey of patients in the Minneapolis-St. Paul area who were treated for heart attacks found that the risk of dying in the three years after the attack is 15% lower than it was in 1985 for women and 25% lower for men.[3] It is recognized, however, that significant practice variations exist across medical care settings and patient groups.

In light of the above facts, treatment for myocardial infarction was identified as a quality improvement project by the Health Care Finance Administration (HCFA). This national Cooperative Cardiovascular Project (CCP) is a quality improvement initiative to assist hospitals and physicians in improving their quality of care delivery to Medicare beneficiaries with acute myocardial infarction, through the measurement of specific quality indicators.

CLINICAL INDICATORS

There are 10 performance measures in the CCP. They include:

1. Reperfusion treatment (thrombolytics or PTCA within 12 hours of presentation to the emergency department)
2. Timing of thrombolytics
3. Timing of PTCA
4. Aspirin during hospitalization
5. Administration of Aspirin within the first day of arrival
6. Aspirin prescribed at discharge
7. Beta Blockers prescribed at discharge
8. ACE inhibitors prescribed at discharge
9. Avoidance of calcium channel blockers
10. Smoking cessation counseling prior to discharge

Note that five of these focus on treatment at the time of presentation to the hospital, and five focus on treatment at time of discharge. In the four state pilot study of AMI indicators, the CCP found that, for all potential candidates, only 18% of patients received thrombolytics, 32% received beta blockers, and 75% received aspirin. It is important to recognize that some of these patients may have had one or more contraindications to such therapies. The baseline measurement in Rhode Island was accomplished by abstraction of 1,360 records of patients who were discharged with a principal diagnosis of AMI (ICD9 code 410) during the period 1994-1996. After exclusions, this resulted in a final study sample size of 1,122 patients.

Table 1
CCP PERFORMANCE MEASUREMENTS
Baseline Period Feb 1994- Feb 1996
Remeasurement Period Sept-1997-Feb 1998

INDICATOR	baseline N=1122	remeasurement N= 577	P-value
Thrombolysis on Admission	57%	70%	0.165
Average Time to Thrombolysis	78 minutes	69 minutes	0.387
Reperfusion on Admission	62%	78%	0.08
Aspirin First Day	58%	51%	0.06
Aspirin during Hospital Stay	83%	93%	<0.001
Aspirin at Discharge	72%	90%	<0.001
Beta Blocker at Discharge	67%	70%	0.754
Ace Inhibitor at Discharge	66%	89%	<0.001
Avoid Ca Channel Blockers in CHF	94%	97%	0.451
Smoking Counseling Given	48%	27%	0.016

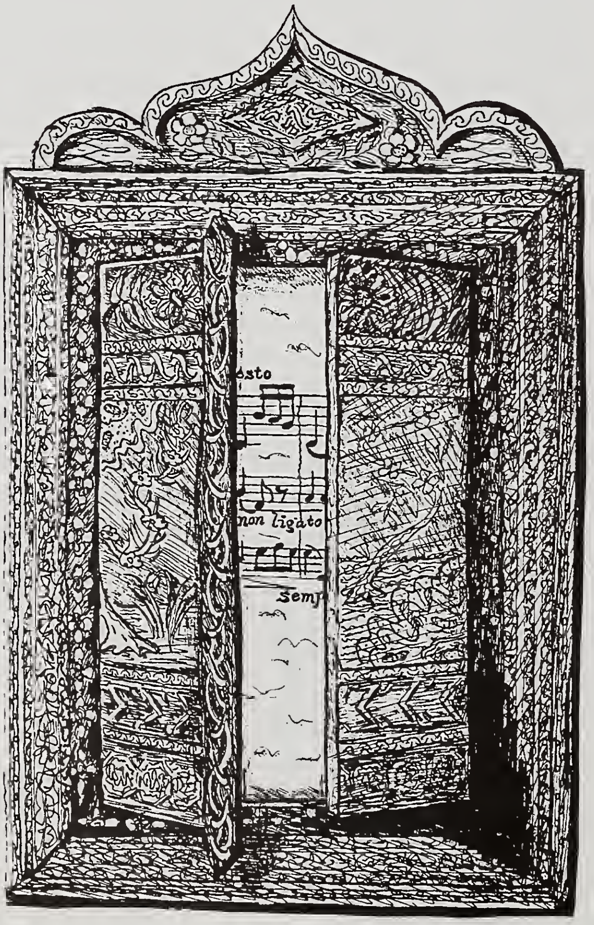
INTERVENTIONS

The baseline performance data were shared with quality improvement representatives from the hospital community in April, 1997. These representatives were asked to share the data with the clinical and administrative leadership at their respective institutions. The ten Rhode Island hospitals, all of whom agreed to collaborate on this project, subsequently implemented a number of improvement measures, focusing on the specific indicators of the CCP.

Some of the steps the different hospitals employed to improve the performance measures on which they focused include such things as:

1. Standing orders initiated or revised
2. Establishment or revision of written protocols
3. Educational pieces given to clinicians (nurses, physicians, nursing assistants, technicians).
4. Monthly monitoring , measurement and feedback loops with health care providers to encourage them to focus on these aspects of care in AMI.

At this time, remeasurement has been completed for these hospitals to assess the effect of the above interventions. There have been 726 patients reviewed by chart abstraction. A final study sample of 577 patients resulted after the application of inclusion and exclusion criteria. The results reveal improvement in most indicators studied (Table 1).



CONCLUSIONS

It appears from preliminary review of the remeasurement data that the project has had areas of significant impact, and some areas with lesser impact. Smoking cessation counseling has been the most disappointing area measured, with an even lower compliance in the remeasurement phase. Some of the poor performance may be due to simple lack of documentation.

Acute MI remains a priority item from HCFA's standpoint and will be a mandatory topic for the Peer Review Organizations in the 6th Scope of Work (every state must do at least one project involving performance improvement in AMI). Future projects may begin to incorporate outcome indicators, although the details have yet to be developed. Progress in these projects will be reported in this column on an ongoing basis.

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Advances in Pharmacology

Selective Estrogen Receptor Modulators: Raloxifene

Amy Rogowski, PharmD, and Brooke Fidler, PharmD candidate

Abbreviations Used:	
HRT	Hormone replacement therapy
SERMS	Selective Estrogen Receptor Modulators
STAR	Study of Tamoxifen and Raloxifene

Since the early 19th century, the life expectancy of women has increased steadily. Most women will now spend a third of their lives in postmenopause. Estrogen deficiency, characterizing the postmenopausal condition, leads to an acceleration of health problems such as osteoporosis and cardiovascular disease. Hormone replacement therapy (HRT) can effectively relieve the climacteric symptoms of menopause, provide cardioprotective benefits, and prevent and treat postmenopausal osteoporosis. However, patients and clinicians are concerned with the reported correlation of HRT to increased risks of breast and endometrial cancer. Fear of this risk and the resumption of menses in some non-hysterctomized women have led to lack of interest in HRT and noncompliance. Recently, a new class of drugs called Selective Estrogen Receptor Modulators (SERMs) have generated interest. SERMs are compounds that mediate estrogenic biological action at some tissues and anti-estrogenic actions at other tissues through differential expression of estrogen-regulated genes as a consequence of receptor binding. The first SERM used was tamoxifen in the prevention and treatment of breast cancer. Tamoxifen was shown to have beneficial estrogen activity on the bone and cardiac system in addition to its beneficial estrogen-blocking activity on breast tissue. However, tamoxifen's endometrial estrogenic effects often leads to endometrial hyperplasia and cancer. A new SERM, raloxifene (Evista®), has been shown to have beneficial estrogen activity in the skeletal and cardiac system and estrogen-blocking activity in both the breast and endometrium.

Table 1.

Major Trials	**Adverse Events**			**Discontinuance due to adverse event**
	Hot flashes	Leg cramps	Other	
601 PMW + osteoporosis Placebo vs. raloxifene (30, 60, 150 mg)[2] 24 months	23% placebo vs 26% (60 mg)	0.7% placebo vs 2.6% (60 mg)	Endometrial thickness: no difference	No significant differences
390 PMW[3] group 1 - placebo group 2 - raloxifene 60 mg group 3 - raloxifene 120 mg group 4 - HRT 6 months	10% group 1 16% group 2 22% group 3 3% group 4	Not reported	Breast tenderness 6% group 1 4% group 2 2% group 3 38% group 4 Vaginal bleeding 5% group 1 5% group 2 3% group 3 45% group 4	Hot flashes 1% group 1 Breast tenderness 2% group 4 Vaginal bleeding 9% group 4
7705 PMW with osteoporosis[4] group 1 - placebo group 2 raloxifene 60 mg group 3 - raloxifene 120 mg Ongoing - interim results at 24 months	5.4% group 1 8.7% group 2	2.4% group 1 5.0% group 2		Not reported

PMW=postmenopausal women

Raloxifene Pharmacology

Raloxifene is FDA-approved for the prevention of osteoporosis in post-menopausal women. It is taken as a 60 mg tablet daily without regards to meals. The drug is >95% protein bound, thus it must be used with caution when administered with other highly protein-bound drugs. Due to extensive hepatic metabolism and enterohepatic recycling, the drug produces higher plasma concentrations in cirrhotic patients. However, there are no specific guidelines for dosage adjustment in patients with hepatic impairment. Raloxifene is excreted in the feces and does not require dosage adjustment in patients with renal impairment. Few drug-drug interactions have been studied, however, cholestyramine significantly reduces the absorption of raloxifene and raloxifene can decrease the prothrombin time when given with warfarin. Raloxifene is contraindicated in pregnancy and in patients with active or past history of thromboembolic events.[1]

The safety profile for raloxifene has been elucidated in postmenopausal women. Cumulative trial data of raloxifene treatment has revealed a relative risk for combined events of deep vein thrombosis and pulmonary embolism similar to that of women taking HRT.[2] Table 1 provides a summary of commonly reported adverse events of raloxifene from published randomized clinical trials.

Ralxofene Trials

Raloxifene was studied in 601 PMW women with and without osteoporosis to determine its effects on bone Mineral Density (BMD), serum lipid concentrations, and uterine endometrium.2 Participants were randomly assigned to receive either 30, 60 or 150 mg raloxifene daily or placebo for 24 months. Calcium supplementation was provided to all participants. All raloxifene treatment groups produced statistically significant decreases in serum bone turnover markers over placebo. Raloxifene at 60 mg/day produced increases of BMD of 2.4±0.4% for the lumbar spine, 2.4±0.4% for the total hip, and 2.0±0.4% for the total body. Low density cholesterol (LDL) decreased (-6.2.0±0.8%, -10.1±1.4%, -14.1±1.6%) with all doses of raloxifene (30mg, 60mg and 150mg, respectively). Total cholesterol also decreased significantly with all study doses of raloxifene. In contrast to HRT, raloxifene treatment did not produce statistically significant changes in high density cholesterol (HDL) or triglycerides. A separate comparative trial of raloxifene and continuous HRT conducted in 390 healthy PMW revealed a similar impact on serum lipid profiles.[3] This trial further explored cardiovascular risk markers such as coagulation factors. Raloxifene significantly lowered fibrinogen by 12 to 14%, unlike HRT, which had no effect. In contrast, HRT significantly decreased plasminogen activator inhibitor-1 whereas raloxifene had no effect.

The on going Multiple Outcomes of Raloxifene Evaluation study (MORE) will provide fracture rate data.[4] Substudy I includes women having osteoporosis in the femoral neck or lumbar spine. Substudy II includes women who have had at least one moderate or two mild vertebral fractures and low BMD, or at least two moderate vertebral fractures regardless of BMD. Interim analysis at twenty-four months has revealed that the relative risk for all participants for having one new vertebral fracture decreased similarly by approximately 50%.

Future

SERMs are being investigated for the primary prevention of breast cancer. Recently, researchers released early results from the Breast Cancer Prevention Trial (BCPT).[5] Women at high risk for breast cancer were enrolled in the BCPT to receive tamoxifen 20 mg/day or placebo. A 45% reduction risk for developing invasive breast cancer for all age groups was reported leading early termination of the trial. However, a trend for greater reduction in breast cancer in women over age 50 was offset by an increased risk for endometrial cancer and thromboembolic events. Researchers are looking to the use of other SERMs, such as raloxifene, in the prevention of breast cancer without detrimental side effects. The Study of Tamoxifen and Raloxifene (STAR) has been announced and is expected to begin early 1999.[6] STAR is designed to enroll 22,000 postmenopausal women 35 or older who are at increased risk for developing breast cancer. Participants will be randomly assigned to receive tamoxifen 20mg/day or raloxifene 60 mg/day for five years. STAR may more fully define the role of raloxifene in breast cancer.

Conclusion

Raloxifene is an effective prevention and treatment option for postmenopausal osteoporosis. Issues will still remain for postmenopausal women suffering from other effects of estrogen deficiency such as urogential atrophy. HRT, as a primary treatment option in osteoporosis, does possess cardiovascular benefits with the downside of possibly increased risk of breast and endometrial cancer. As of yet, there are no comparative trials between raloxifene and nonhormonal drug options, such as alendronates, for the prevention and treatment of osteoporosis. Therefore, it is beyond the scope of this article to comment on the comparative efficacy between the two drugs. The SERMS may be a therapeutic choice for PMW with hypertriglyceridemia, history of gall bladder disease, or a family history of breast cancer for the prevention or treatment of osteoporosis. The role of raloxifene, as well as other future SERMS in development (i.e. droloxifene), may be defined by future trials.

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PUBLIC HEALTH BRIEFING

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

Public Health Alert: Follow-up on Palpable Lesions of the Breast with Normal Mammogram Results

Donald K. Perry, MPA, and Dolores DiOrio, RN, MEd

BACKGROUND

Funded through the national Centers for Disease Control and Prevention (CDC), the Rhode Island Women's Cancer Screening Program (WCSP) in the Department of Health provides no-cost Pap tests and screening mammograms to high risk women (i.e., uninsured and low income) throughout the State. The program has provided screening services as well as necessary follow-up diagnostic procedures since 1995. During that time, the WCSP has also worked with the medical community to develop and promulgate health care guidelines related to the early detection and diagnosis of breast and cervical cancer. These guidelines have been enthusiastically received and are aimed at improving the quality of care and health outcomes not just for high risk populations but for all Rhode Island women.

FINDINGS: WOMEN'S CANCER SCREENING PROGRAM DATA

Despite the widespread dissemination of the guidelines, there appears to be confusion over the proper follow-up for palpable lesions and other abnormal findings from clinical breast exams. Program data reveal that a radiologist's report of a normal screening mammogram (i.e., negative, benign, probably benign) may deter further diagnostic workup of a palpable mass. This is inadvisable and may compromise a patient's health if cancer is present.

Abbreviations Used:

CDC	Centers for Disease Control and Prevention
FNA	fine needle aspiration
NBCCEDP	National Breast and Cervical Cancer Early Detection Program
WCSP	Women's Cancer Screening Program

DISCUSSION: EXPERT RECOMMENDATIONS

In a national article published earlier this year by the American Cancer Society,[1] Dr. Blake Cady, et. al., stressed that "a normal mammogram at any age does not eliminate the need for further evaluation of a palpable suspicious mass." The authors define such a mass as "solitary, discrete, and firm or hard; it may or may not be fixed to adjacent tissue." This issue is so critical that the CDC reprinted and disseminated the article to public health agencies across the country. When consulted recently on the WCSP data described above, Dr. Cady offered this additional admonition:

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Table 1
Timeliness and Adequacy of Follow-up Algorithm For Breast Cancer Screening

Principles

- A diagnostic work-up must be planned whenever there is an abnormal clinical breast exam (CBE), and/or when the screening mammography is 'suspicious abnormality,' 'highly suggestive of malignancy,' or 'assessment incomplete.'
- Whenever a diagnostic work-up is planned, a final diagnosis must be recorded for follow-up to be considered adequate.
- Whenever a diagnostic work-up is planned, the time from screening mammogram or CBE (whichever occurs first) to final diagnosis must be no more than 60 days.

Clinical Breast Exam	Mammogram	Adequate Follow-up Diagnostic Procedures	Comment
Normal	Negative, Benign finding, Probably benign - short-term follow-up	No work-up need be planned. If work-up is planned, at least one diagnostic procedure must be done, and a final diagnosis recorded.	
Abnormal	**Negative, Benign finding, Probably benign-short-term follow-up, Assessment incomplete**	**One or more of the following: Repeat breast exam/surgical consult Ultrasound, Biopsy/lumpectomy Fine needle/cyst aspiration**	**Mammogram or additional mammographic views as only procedure is not adequate.**
Abnormal	Suspicious abnormality, Highly suspicious of malignancy	Biopsy/lumpectomy Fine needle/cyst aspiration	
Normal	Suspicious abnormality	Repeat breast exam/surgical consult Ultrasound, Biopsy/lumpectomy Fine needle/cyst aspiration	
Normal/ Abnormal	Highly suspicious of malignancy	Biopsy/lumpectomy or Fine needle/cyst aspiration	
Normal	Assessment incomplete	Additional mammographic views or Ultrasound	

Abnormal and/or 'Suspicious abnormality' or 'Highly suspicious of malignancy' or 'Assessment incomplete' - If no final diagnosis is recorded, then work-up is Inadequate

Source: The Centers for Disease Control and Prevention, National Breast and Cervical Cancer Early Detection Program

"One of the most dangerous situations in breast cancer management today is when a woman with a breast mass has a normal mammogram. Such women with palpable masses must be resolved based on the clinical setting. The clinical mass needs specific evaluation by physical examination and perhaps other studies such as ultrasound or even fine needle aspiration (FNA). A high proportion of medical malpractice claims revolve around this very issue and the possibility of delaying the diagnosis of breast cancer. All physicians must remember that mammography is specifically *not* to evaluate what can be felt but only to evaluate by screening what is *not* felt."

Reflecting these concerns, the National Breast and Cervical Cancer Early Detection Program (NBCCEDP) at the CDC has developed protocols on the timeliness and adequacy of follow-up care for breast cancer screening. Reprinted in Table 1, these protocols have been issued to the NBCCEDP programs in all 50 states, including the Rhode Island WCSP, as well as several territories and Indian nations.

The Rhode Island Department of Health, WCSP staff, and collaborating Rhode Island medical care providers hope that this alert will resolve any confusion or misunderstanding regarding the diagnostic workup for palpable breast lesions. As partners, we can continue to assure the highest quality of cancer care. Please direct any questions, comments, or concerns to Dolores DiOrio of the WCSP staff at (401) 222-1394, x127.

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Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health
Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report
Provisional Occurrence Data from the Division of Vital Records

Vital Events

	Reporting Period		
	July 1998	**12 Months Ending with July 1998**	
	Number	**Number**	**Rates**
Live Births	1,234	13,339	13.5*
Deaths	774	9,806	9.9*
Infant Deaths	(12)	(103)	7.7#
Neonatal deaths	(10)	(80)	6.0#
Marriages	705	7,601	7.7*
Divorces	238	3,203	3.2*
Induced Terminations	451	4,879	365.8#
Spontaneous Fetal Deaths	59	959	71.9#
Under 20 weeks gestation	(47)	(883)	66.2#
20+ weeks gestation	(12)	(76)	5.7#

* Rates per 1,000 estimated population
# Rates per 1,000 live births

Underlying Cause of Death

	Reporting Period			
	January 1998	**12 Months Ending with January 1998**		
	Number (a)	**Number (a)**	**Rates (b)**	**YPLL (c)**
Diseases of the Heart	288	3,216	324.8	3,664.5
Malignant Neoplasms	209	2,489	251.4	7,219.5
Cerebrovascular Diseases	61	677	68.4	717.0
Injuries (Accident/Suicide/Homicide)	31	318	32.1	6,384.0**
COPD	60	439	44.3	322.5***

**Excludes two deaths of unknown age ***Excludes one death of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.
(b) Rates per 100,000 estimated population of 990,225
(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

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NINETY YEARS AGO

[JANUARY, 1909]

In honor of the centennial of the births of Charles Darwin and Abraham Lincoln, the Brown University Departments of Biology and Botany planned on February 12 to "unite with the medical profession of Providence in fraternal recognition of Darwin's claim to remembrance" for "review of the century's progress in natural science."

Edward E. Pierce, MD, in an article entitled "Vomiting," described the symptoms from his own experiences ("and hence holding to originality, so far as anything in medical literature may be considered purely original"). He noted a vomiting center in the medulla oblongata, perhaps connected to the various bodily organs. He noted too individuals' differences: "some people vomit at the odor of vomitng; other people hardly ever mind." Among the causes, Dr. Pierce cited injuries to the head, diseases of the brain, sensory causes (including "thought of blood"), diseases of the liver, seasickness, and homesickness.

Fenwick G. Taggart, MD, urged colleagues ("Some Peculiarities in the Stools of Young Children) to examine carefully the stools of young patients, using litmus paper and a spatula ("No chemical laboratory or microscope is necessary"). He noted that breast-fed babies' stools rarely presented a medical concern.

V.L. Raia, MD, presented 3 cases of glaucoma. In one, a 36 year-old Englishman had lost his left eye 11 years before to glaucoma. Five years ago, when his right eye was affected, physicians at Rhode Island Hospital performed an iridectomy, followed by a posterior sclerotomy. Last year, after a relapse and more surgery, the patient's vision again blurred, and he went totally blind.

At a meeting of the Rhode Island Medical Society, Dr. Arthur Harrington read a paper on State Care of the Insane. Dr. Blumer concurred, calling the conditions "a discredit and a disgrace."

FIFTY YEARS AGO

[JANUARY, 1949]

This issue featured a symposium on cancer.

George W. Waterman, MD, FACS, and Sumner I. Raphael, MD, discussed cancer of the uterus, which "stands about equally with breast cancer as a cause of death in the female population." They stressed the benefits of a Cancer Control Program, which emphasized "lay education in early signs and symptoms," professional education in the latest methods of diagnosis, cancer detection clinics for well persons, a cancer registry, and effective treatment.

Herman C. Pitts, MD, traced the history of the Cancer Detection Clinic to the 1937 clinic at New York Infirmary for Women and Children, where women could come twice a year for a thorough exam "directed particularly against cancer." The popularity of this clinic spurred the American Cancer Society to sponsor additional ones. In 1946, with the approval of the RI Medical Society, the state's first Cancer Detection clinic was started at Rhode Island Hospital. Physicians were paid from state funds; appointments were made in the State House. The Rhode Island Cancer Society underwrote administrative costs. In two years, 4 more clinics began - 2 at Rhode Island Hospital, 1 at Memorial, 1 at Newport. The clinics did not interfere with physicians' practice, since women received no treatment. Dr. Pitts foresaw the end of the clinics: "If and when each doctor's office becomes truly a Cancer Detection clinic, then these other clinics....can be closed and the responsiblity for the health of our people placed entirely where it belongs - in the hands of the conscientious practicing MD."

An editorial on "British Socialism" lamented the "stampede for 'free care.' Free false teeth are also in great demand. It is reported that most dentists pull each other's teeth, which they formerly did as a pleasant, neighborly mutual service, and then send bills to the government for payment..."

TWENTY FIVE YEARS AGO

[JANUARY, 1974]

Speaking before the Hampden District Medical Society, Senator Wallace I. Bennett (Utah), a ranking minority member of the Senate Finance Committee, urged opponents of Peer Standard Review Organizations (PSROs) to work with the government for changes. Conceding the "snail's pace" of implementation, Bennett warned of the public relations impact of opposition: "I would foresee headines.. 'Doctors refuse to review medical care.'" (The Journal reprinted the text of his remarks).

Arvin S. Glicksman, MD, and Banice M. Webber, MD, ("Recent Trends in Oncology") called for the involvement of multiple medical specialties, and noted the progress in Hodgkin's Disease and acute lymphomatic leukemia.

Francis L. McNelis, MD, attributed increased incidence of carcinoma-in-situ of the larynx to better diagnosis with the use of microlaryngoscopy. It was the diagnosis in 12 cases at Rhode Island Hospital from 1968-70, with 8 cases treated with full tumoridical dosage ("Irradiation was felt to be the treatment of choice.") Today "It is now our policy to avoid irradiation ... unless the lesion is ...widespread."

An editorial discusses generic versus brand-name drugs, concurring with the AMA, the ACP, the American Academy of Family Physicians, and the Pharmaceutical Manufacturers of America against substitution: "What the physician requires of a drug is therapeutic equivalence, which is not discernable by the pharmacist."

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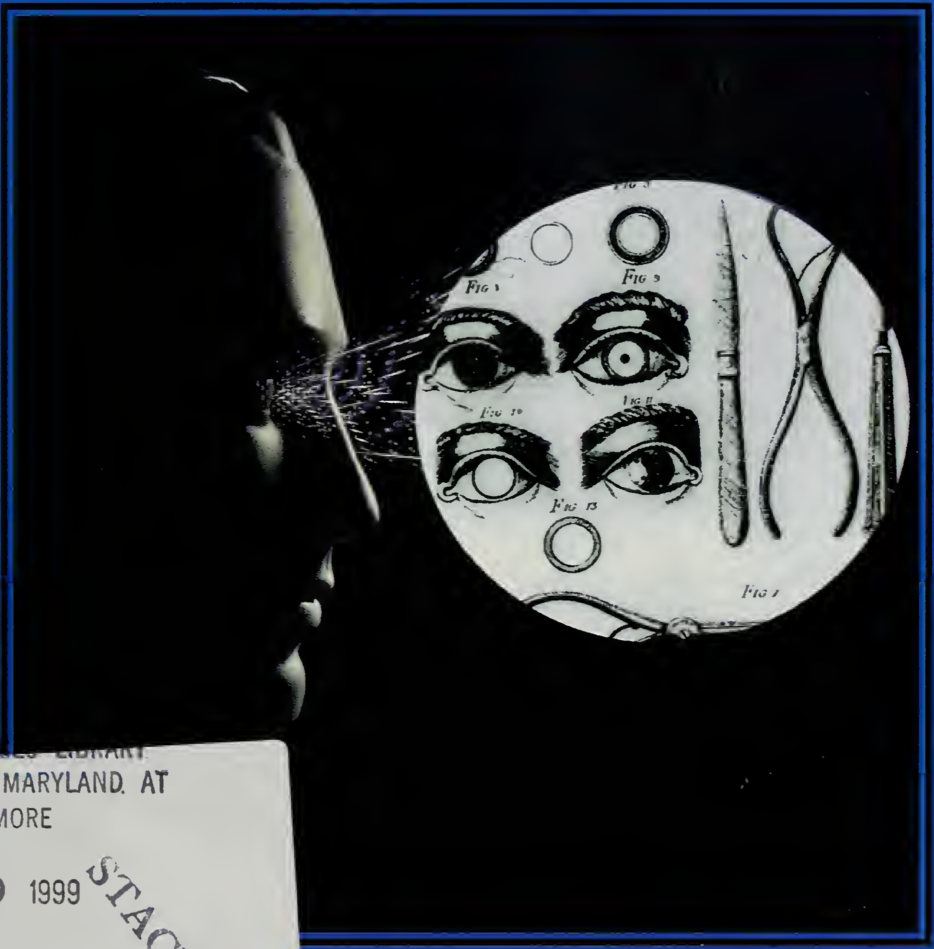

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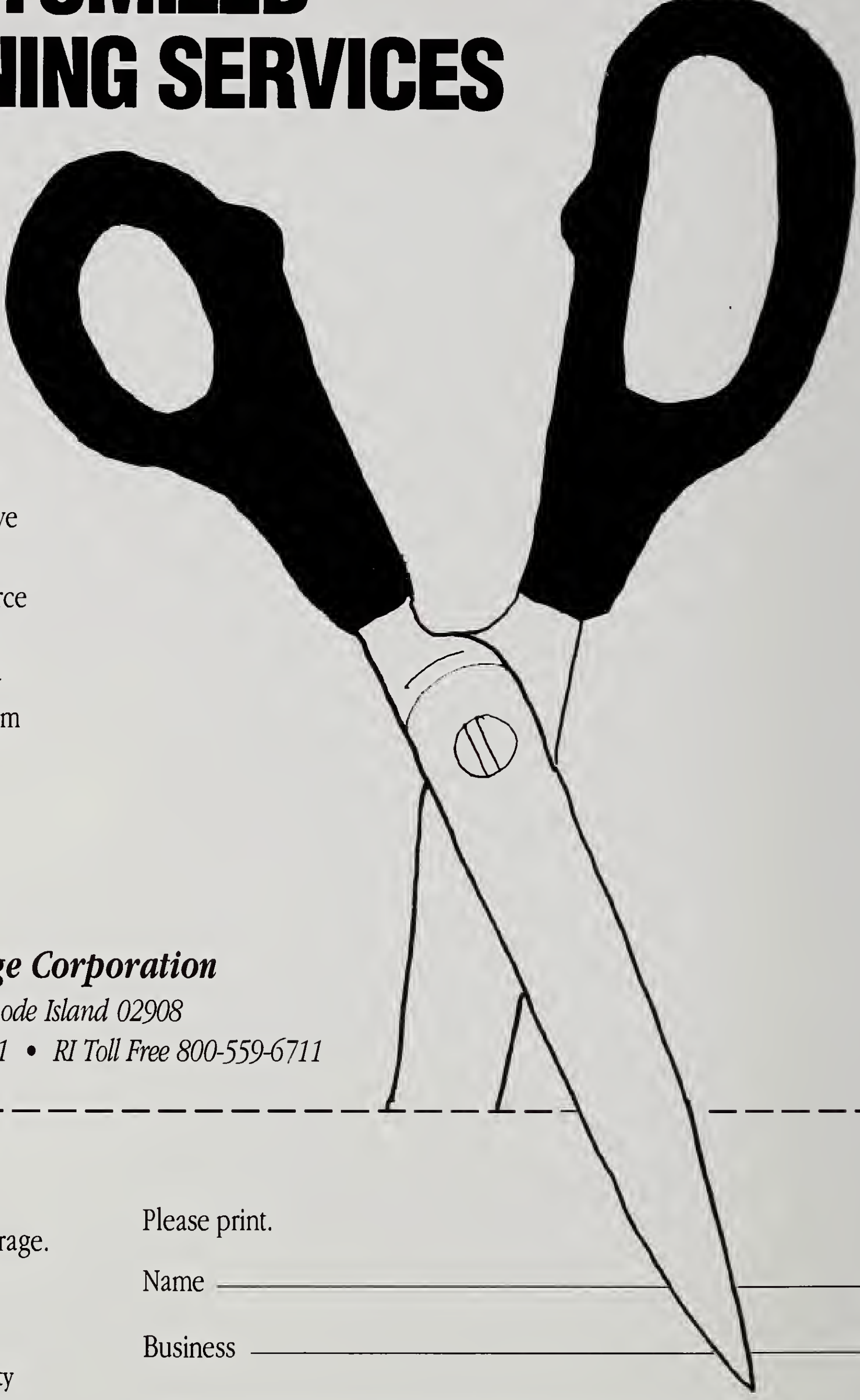

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Cover: DIAGRAM BY DEQUENGSY showing placement of watch crystal into a scarred cornea. (From Casey TA & Mayer DJ, *Corneal Grafting*, Philadephia, WB Saunders, 1984).

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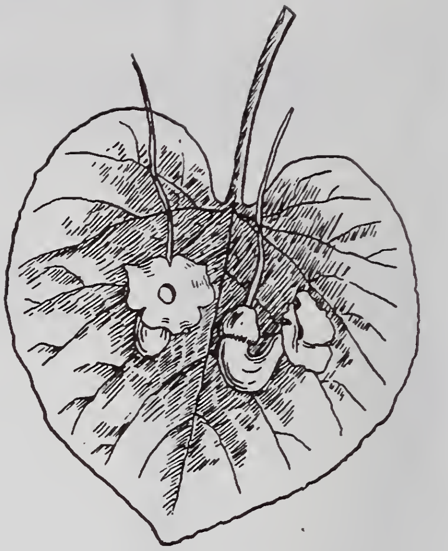
COMMENTARIES

New Perspectives on Old Diseases



In the span of only a decade, the practice of ophthalmology has changed radically. Because of the dramatic success of outpatient cataract surgery, ophthalmic surgery inpatient beds and eye hospitals are virtually empty. But this success has also had an unfortunate consequence: it has moved ophthalmologists out of the hospital setting, leaving us as strangers who visit the hospital for consultations and attendance at selected rounds. The result is that ophthalmologists and non-ophthalmic physicians rarely get a full understanding of each other's specialties.

My intent in this pre-millennium issue is to focus on the ways in which ophthalmologists currently deal with five common eye problems. All should be familiar to most physicians, since each has been recognized and treated for over a century. Three of the five deal with ophthalmic surgery. My goal here is, in part, to allow non-ophthalmologist physicians — not only the internists and generalists who evaluate our patients preoperatively, but also the anesthesiologists who monitor them intraoperatively — to understand how different the practice of eye surgery is now from what it was, even at the beginning of the decade. I believe this understanding will help us better manage our ophthalmologic patients together.

I also trust that this issue of *Medicine & Health/Rhode Island* will encourage non-ophthalmologists to take a more careful (perhaps even more favorable) look at ophthalmic surgical practices. In an era when cost-containment, easy access to care, and patient safety are often highly valued but mutually exclusive goals, ophthalmologists have been successful in balancing all three. I hope the articles will illuminate elements of outpatient surgery philosophy which non-ophthalmic surgeons will find useful.

Consider the patient undergoing cataract surgery, as discussed by Dr. Robert Bahr. The surgery is bloodless and sutureless and is performed using just topical anesthetic. It hardly seems any more invasive than having a cavity filled in the dentist's office (except for the intravenous line the anesthesiologist places). Given this type of surgery, how much preoperative medical care and evaluation should we ophthalmologists be asking our colleagues to provide? How much intraoperative systemic monitoring should we ask anesthesiologists to deliver? There are no clear-cut answers to these questions, but they deserve serious consideration.

Dr. Lory Snady-McCoy provides another example of how we have moved towards less invasive procedures for common eye conditions. Dr. Snady-McCoy discusses pneumatic retinopexy, in which retinal detachment is repaired in an office setting with no incision and little more than topical anesthetic. Certainly this is a far cry from the 5-day inpatient stay common even 10 years ago.

My own article on corneal transplantation highlights the dramatic changes in the availability of this vision-saving procedure. In less than 25 years, corneal transplantation has evolved from a rarely done operation with mediocre results to a regularly scheduled surgery with a high success rate.

The previous authors deal with ocular surgical procedures that have little direct bearing on general medical problems. In contrast, Drs. Thanos Zervos, William Tsiaras and Deborah Murphy address other prevalent ophthalmologic entities that are medically managed and have significant relevance to general medicine.

Drs. Zervos and Tsiaras deal with auto-immune diseases and their effects on the eye. Managing these multisystem diseases requires coordinated efforts between ophthalmologists and various medical specialists to ensure good patient care. To be successful, all the physicians involved must have a common understanding of these complicated disease processes. I am optimistic that this paper will help in this regard.

Finally, Dr. Deborah Murphy addresses an ophthalmic condition we all thought we understood and could treat: glaucoma. Watching some glaucoma patients go blind "before our very eyes" forces us to re-evaluate this disease and how we might treat it differently.

Prompted by research in other fields such as stroke management, ophthalmic researchers are evaluating the role of apoptosis and neuro-protective mechanisms in the pathogenesis of glaucoma. Conceivably our approach to this entity in the next century may have very little to do with how we have managed it in the past.

I am confident that the reader will find that these diverse papers on "old" ophthalmologic problems will provide fresh information and new insights.

– Elliot M.Perlman, MD

Elliot M. Perlman, MD, is clinical assistant professor of surgery, Brown University School of Medicine and director of the Corneal Service, Brown University and Rhode Island Hospital. He is on the staff of Rhode Island Hospital and Miriam Hospital and is in private group practice.

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Eye Witness Accounts of the Great Plague

History, it is reliably said, moves inexorably forward whether or not there are observers to document its occasionally sordid progress. Probably true; but, like verifying the sound of a tree falling in an unpopulated forest, it is difficult to prove.

Before the advent of the electronic media; before the television screen portrayed the grim features of human mishap in florid color; before journalism brought instant news to each neighborhood precinct, there were writers to convey what they had seen. The writings of compulsive archivists such as Herodotus, Xenophon, Josephus and others have left us with a glimpse of prejournalistic history.

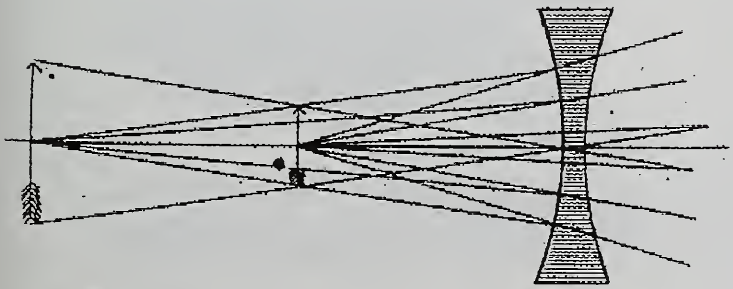
In more recent centuries, descriptions of current events were often captured in the private writings of those who felt compelled to record their daily experiences in secret diaries or daily journals. London's awesome calamities of 1665 are a case in point. Much of what is known of the Great Plague of 1665 as well as the texture of life during those perilous months is preserved in the diaries of contemporaries who documented the appalling events which befell the British capitol.

There was, for example, Samuel Pepys [1633-1703] who held a succession of minor court positions and was eventually elected to the House of Commons. He remained in London during the plague. His encoded diary, spanning the years 1660-1669, when finally deciphered, proved to be an amazingly informative, verbose, gossipy document giving vivid details of 17th Century life ranging from the foods consumed to the most intimate idiosyncracies of his peers.

John Evelyn [1620-1706], compulsive traveler, patron of the arts, royal spy and sometime fiscal officer of the British Navy, maintained a personal diary from 1640 to 1706. This, too, was a prolix journal, though richer in political than interpersonal gossip. Pepys and Evelyn might have been aghast at the thought that their intimate diaries were to be avidly read by generations of college students.

Daniel Defoe [1659-1725] published a densely detailed journal of the plague years portraying London during its grim travail. Defoe was only six years old when the plague struck. His realistic account of the plague, he claimed, was derived from a diary maintained by his uncle, Henry Foe.

England had experienced prior plague epidemics in 1602 and 1625, by coincidence, coronation years for James I and Charles I. A resurgence of the plague was therefore anticipated in 1660 when Charles II was to be enthroned. The plague, however, did not appear until 1665 and was heralded, as predicted by soothsayers, by a feverish comet in the sky.



> *Defoe*: "A blazing star or comet appeared for several months. The old women remarked that this comet passed directly over the city; that it was plain that it imported something peculiar to London."

> *John Gadbury* [a local astrologer]: "Such comets of leaden, envious, pale, ashy color are saturnine and they denote that there shall happen in the world many pernicious evils as famine, absolute destruction of all things and plague."

> *Dr. George Thomson* [a prominent London physician]: "That comets do portend some evil to come upon mortals is confirmed by long observation and sad experience, as likewise phenomena of new stars, battles fought, and coffins carried through the air, howlings and groans heard about churchyards, also raining of blood."

> *Pepys*: "And so to the coffee house where great talk of the comet seen in several places; and among our men at sea and by my Lord Sandwich to whom I intend to write about it tonight. Thence home to dinner."

The desolate streets of London during the height of the plague, as seen by contemporaries:

> *Defoe*: "Death now began not to hover over everyone's head only, but to look into their houses and chambers and stare into their faces. Many consciences were now awakened and many a penitent confession was made of crimes long concealed. An abundance of quacks also died who had the folly to trust to their own medicines."

> *Defoe*: "They fled in such a manner that I began to think there would be really none but magistrates and servants left in the city. The court also removed early in June and went to Oxford where it pleased God to preserve them. London might well be said to be all in tears. The voice of mourning was heard truly in the streets. The shrieks of children and women at the windows. It was enough to pierce the stoutest heart. The inns of court were all shut, nor were very many of the lawyers in the temple to be seen. Since everybody was now at peace, there was no occasion for lawyers."

> *Defoe*: "The apprehensions of the people were likewise strangely increased by the error of the times and the people were more addicted to prophesies and astrological conjurations, dreams and old wives' tales than ever before."

Evelyn: "Came home, there perishing nearly 10,000 poor creatures weekly. I went along the city from Kent St to St. James, a dismal passage, and dangerous to see how many coffins exposed in the streets now thin of people."

Pepys: "I could walk Lumber St and not meet 20 souls from one end to the other; my very physician who undertook to secure me against any infection died himself of the plague; till the nights, much lengthened, had grown too short to conceal the burials of those that died the day before. Grass grows up and down Whitehall Court and nobody but poor wretches in the streets."

By August London was no longer recognizable as the capitol of a great nation. The royalist emigres who had brought great wealth to the city were again scattered by an enemy more potent than Cromwell. The plague subsided by late autumn, lingered somewhat through 1669 and then was no more. Two mysteries remained: why the plague arrived; and, then, why it departed.

– Stanley M. Aronson, MD

A New Editor for the Journal

This medical journal has been published without interruption for over one hundred years, first as the *Providence Medical Journal,* then in 1917 as the *Rhode Island Medical Journal,* and now under its present, more comprehensive, name. Its editors have tended to remain at their posts for lengthy intervals. And our present editor, Stanley M. Aronson, MD, has maintained this tradition. With this issue he will have completed ten years, one hundred and twenty journal issues, in this volunteer position. And, in his judgment, this seemed to be an eminently appropriate and propitious time to retire.

The Editorial Board, Chaired by Edward Feller, MD, is now deeply pleased to announce the appointment of our new editor: Joseph H. Friedman, MD.

Dr. Friedman was born in New York City and received his baccalaureate degree, majoring in mathematics, from the University of Chicago. For the following two years he worked in the Peace Corps as a mathematics instructor in Bimbilla, Ghana. He then returned to the States, earning a master's degree in mathematics from Washington University before entering Columbia University's College of Physicians and Surgeons for his MD degree.

A year of internship in internal medicine at New York's Mount Sinai Hospital preceded his residency training in neurology at the Neurological Institute of Columbia University. In 1982, now a Board-certified neurologist, Joe Friedman came to Rhode Island to assume the position of Chief, Division of Neurology, Roger Williams General Hospital and an assistant professorship at the Brown University School of Medicine.

During the next 16 years, Joe Friedman's career in this state has blossomed. He is now professor of clinical neurosciences at Brown, Chief of the Division of Neurology at Memorial Hospital of Rhode Island, consulting neurologist to the majority of this state's hospitals, both public and private, and is one of this nation's leading authorities on abnormal movement disorders, particularly Parkinson's disease. He has published extensively with over 189 papers, text chapters and abstracts in the medical literature. And during the last year, he has been learning the technicalities of this journal as its associate editor-in-chief.

Joe Friedman is married to Susan Mates, MD, and lives in Barrington with their three growing children.

The Editorial Board is deeply pleased that this medical journal, sponsored jointly by the Rhode Island Medical Society, the Brown University School of Medicine and the Rhode Island Department of Health, is to be supervised by a person of such competence, literary ability and clinical experience. We look forward to at least a decade of editorial service from Joe Friedman as ***Medicine & Health/Rhode Island*** enters the next century.

– Edward R. Feller, MD
– Stanley M. Aronson, MD
– Joan Retsinas, PhD

New Perspectives in Cataract Surgery

Robert L. Bahr, MD

Abbreviations Used:

BAT	brightness acuity test
FDA	Food and Drug Administration
PMMA	polymethyl methacrylate

There has been a marked transformation in all aspects of cataract surgery since the 1960s. For example, the indications for cataract surgery have changed considerably. Rather than tolerating diminished vision while waiting for their cataracts to "ripen," as we used to recommend, patients can now anticipate surgical restoration of their sight when their quality of life and visual function are first compromised. A dramatic improvement has also occurred in our method of optical correction: instead of having their vision limited by thick, heavy, distorting spectacles, patients often can function without any glasses because of prosthetic intraocular lenses. Postoperative care has changed substantially as well. Patients are discharged from ambulatory surgical facilities with already improved vision within twenty minutes of the completion of surgery in comparison to spending several postoperative days hospitalized with bedrest and with their heads immobilized by sandbags.

This paper will explore the current status of cataract surgery with respect to indications for cataract extraction, current surgical techniques and perioperative considerations, and postoperative quality of life.

INDICATIONS FOR SURGERY

Cataract is a major cause of visual handicap in the United States. In the Beaver Dam eye study visually significant cataracts were noted in the worse eye of 3.9% of men and 10% of women from 55 to 64 years of age, 14.3% of men and 23.5% of women between 65 and 74, and 38.8% of men and 45.9% of women over 75 years of age.[1] More than two million cataract surgeries are now performed annually. It is the surgical procedure most frequently performed on Medicare beneficiaries.

In otherwise healthy eyes, between 96% and 99% undergoing cataract surgery with posterior chamber intraocular lenses achieved 20/40 or better acuity in many FDA studies. (Figure 1) The foremost considerations in the decision to perform surgery today are the patient's visual requirements for daily life. For example, the ability to see well enough to drive a car is extremely important to active, independent individuals. Acuity of better than 20/50 in one eye is required by law in most states in order to drive.

Certain types of cataract, such as posterior subcapsular or cortical opacities, cause more significant vision problems in bright sunlight or with glare induced by headlights at night, making driving more difficult. In order to assess these factors in the ophthalmologist's office, the patient's best-corrected visual acuity is first determined and then the effect of brightness or glare upon vision is quantified using a Brightness Acuity Test (BAT) or contrast sensitivity test. These simple optical devices shine extra light near the patient's eyes to simulate external glare.

The most common type of cataract associated with the aging process is nuclear sclerosis, which is characterized by increased central lenticular density and brunescence, accompanied by a myopic refractive shift. Thus the patient experiences a gradual improvement in perceived near-vision coupled with a decrease in distance acuity. Many geriatric patients are very tolerant of a decrease in distant acuity if they can continue to read well. Thus cataract surgery is often not necessary in a sed-

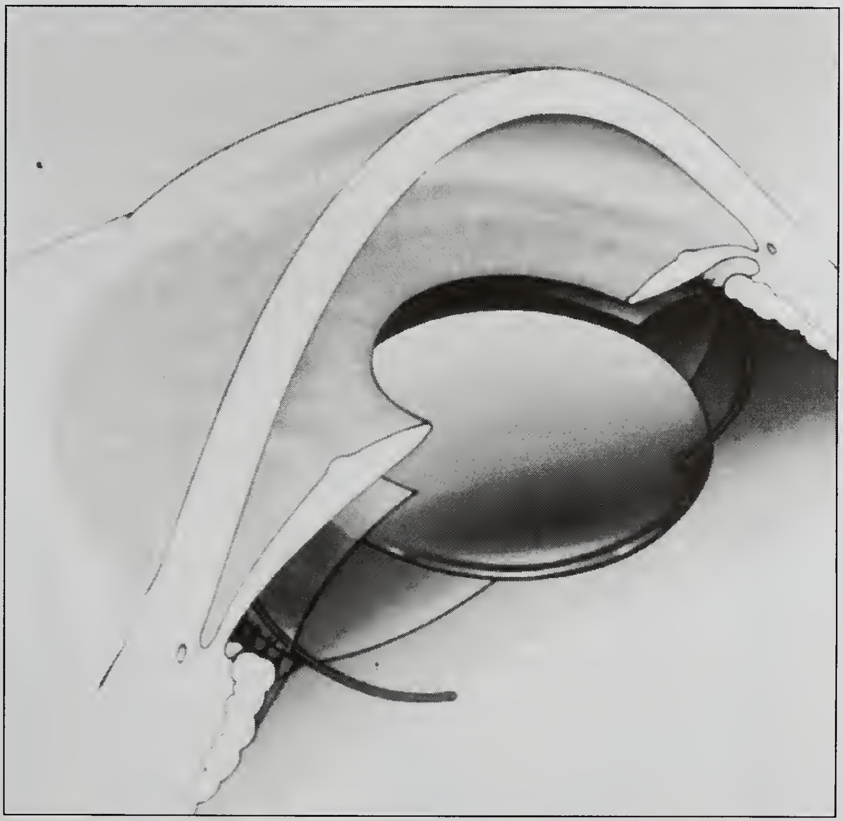


Figure 1: The posterior chamber intraocular lens is placed posterior to the iris and within the capsular bag which previously contained the human lens.

entary elderly patient who can function well in his home with 20/60 or 20/ 80 distance acuity. It is important to remember that reading or watching television may be the primary pleasurable pastimes of patients with severely debilitating or terminal medical conditions. The restoration of these visual pursuits can enhance their lives dramatically.

During the evaluation of a cataract patient, the ophthalmologist searches for evidence of other ocular pathology that could limit the expected benefits of cataract surgery, such as glaucoma, macular degeneration, or diabetic retinopathy. If such conditions exist, the ophthalmologist must determine to what degree the potential visual benefit from cataract extraction will be affected, so that the patient does not have false expectations. In the face of advanced age-related macular degeneration, for example, a cataract would have to be dense enough to limit peripheral vision to merit consideration for removal. With mild macular degenerative changes consistent with 20/30 acuity, the removal of a relatively mild cataract could well allow a patient to achieve enough clarity to drive legally. Patients with occupations requiring extremely precise vision (such as diamond cutters or ophthalmologists) might require the removal of mildly cataractous lenses in order to maintain their occupational proficiency.

In children, cataract surgery is indicated if the lenticular opacity is sufficiently dense to cause amblyopia. In infants with dense cataracts, surgery is indicated as early in the neonatal period as it can be safely performed in order to prevent profound amblyopia and nystagmus. Intraocular lens implantation has become commonplace in all but infants under one year of age and has alleviated problems with fitting aphakic spectacles or contact lenses postoperatively. The major challenge in sight restoration after pediatric cataract extraction is to prevent amblyopia. This goal is greatly facilitated by providing the child with a relatively normal uncorrected postoperative refractive error thanks to the implanted lens.

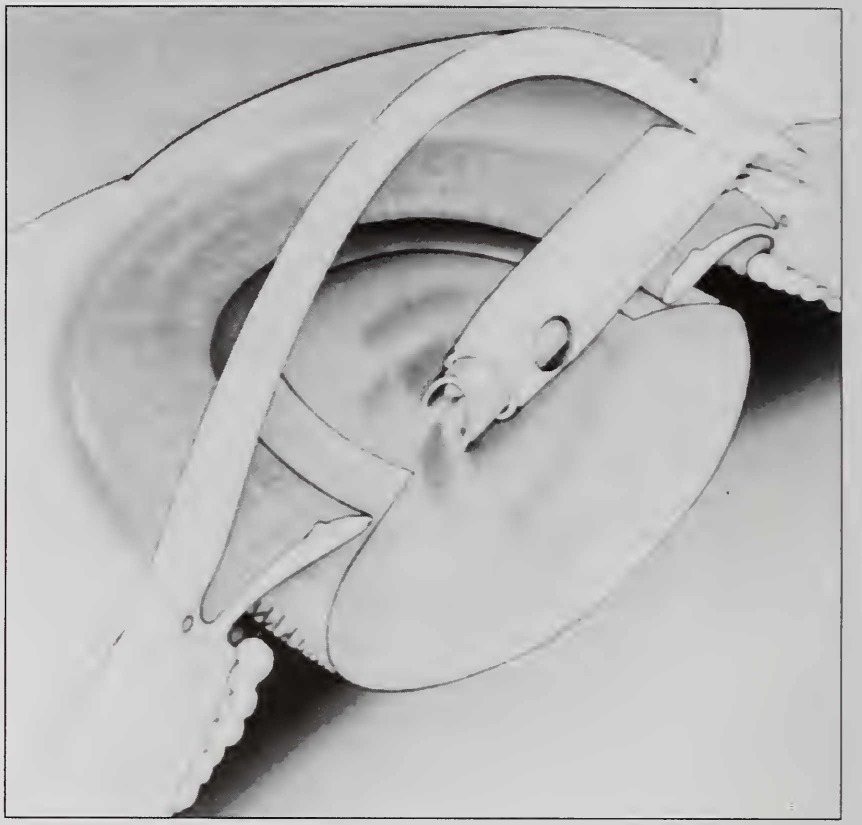


Figure 2: Phacoemulsification is a technique where an ultrasonic needle is used to fragment and aspirate the lens nucleus within the anterior chamber.

GENERAL MEDICAL CONSIDERATIONS

The general medical status of the patient must be considered. Elderly patients, who often have multiple serious medical conditions, make up the majority of cataract surgery patients. Since the surgery is commonly performed employing local topical anesthesia drops (usually with additional anesthetic instilled into the anterior chamber as well, termed intracameral administration), even relatively poor general surgical candidates can undergo cataract surgery without significant compromise of their systemic status. With the help of preoperative counseling, patient anxiety and stress related to the surgery is minimized, and, as a result, so is the need for sedation. The use of topical anesthesia avoids systemic anesthetic absorption and the need for epinephrine to extend the duration of anesthesia. Unless contraindicated, minimal mild sedation (through a small intravenous line) usually accompanies the use of topical anesthesia. Topical anesthesia can be employed in any cooperative patient who can fixate on the microscope light and follow simple commands. Systemic risks and local ocular risks are almost non-existent. In contrast, when a local regional block is performed by injecting anesthetic around the eye (peribulbar anesthesia), there is a small risk of globe perforation or inadvertent intravascular medication delivery with systemic effects. With a retrobulbar block, in addition to the risks of peribulbar anesthesia noted above, inadvertent intrathecal anesthetic administration via the optic nerve sheath can very rarely cause respiratory arrest.

Since the patient is covered by drapes during surgery, the potential of inducing claustrophobia must be considered. A special clear drape, allowing the patient to see with the unoperated eye, usually avoids serious anxiety. Surgery is performed with the patient in supine position; therefore, it is important to have an awareness of the individual's ability to lie flat. Patients with pulmonary or cardiac conditions requiring the head to be elevated can do so. Placing the patient in mild reverse Trendelenberg position usually allows both the patient and ophthalmologist to be comfortable.

The rare situation where the patient's head has to be markedly elevated used to be awkward in the past when the surgical incision was routinely performed superiorly. Now that surgery is commonly performed employing a temporal corneal incision, elevation of the head of the operating table does not compromise the approach as much.

When general non-ocular surgery is contemplated, the surgeon must be aware of any systemic conditions that might predispose the patient to serious bleeding problems. It is important to recognize that surgery on the eye for cataract, in contrast, is quite different from surgery performed on most internal organ systems, since cataract surgery does not involve any significant vascular structures. The only significant hemorrhagic complication of cataract surgery is choroidal hemorrhage, a rare devastating complication associated with vascular instability during periods of hypotony during surgery. Bleeding caused by creating an incision is not a significant concern during cataract surgery. In the case of a clear corneal incision approach no vessels are incised. As a result it is **not** necessary to discontinue Coumadin anticoagulation or the administration of aspirin prior to cataract surgery. Usually the only complication of continuing these medications is the presence of a postoperative subconjunctival hemorrhage from incidental contact of an instrument with a conjunctival vessel. If a patient's medical condition truly requires anticoagulation, the risk of cerebrovascular accident or death with its discontinuation outweighs the fact that a rare choroidal hemorrhage might be worse in the face of anticoagulation. Since severe hypertension is associated with an increased risk of choroidal hemorrhage, efforts should be made to normalize blood pressure prior to considering surgery. Although an attempt should be made to optimize the control of diabetic patients in order to optimize healing, elevated blood sugar is not a contraindication to the surgery. Hypoglycemia at the time of surgery should be avoided. Conditions which elevate orbital pressure or orbital venous pressure increase the potential for increased pressure on the globe and can cause intraocular structures (iris or vitreous) to prolapse from the cataract incision during surgery. Thus thyroid ophthalmopathy or carotid-cavernous sinus shunts should be treated prior to considering cataract surgery. Patients on diuretics should hold these medications until immediately after surgery in order to avoid a full bladder in the midst of surgery.

A dramatic improvement has also occurred in our method of optical correction: instead of having their vision limited by thick, heavy, distorting spectacles, patients often can function without any glasses because of prosthetic intraocular lenses

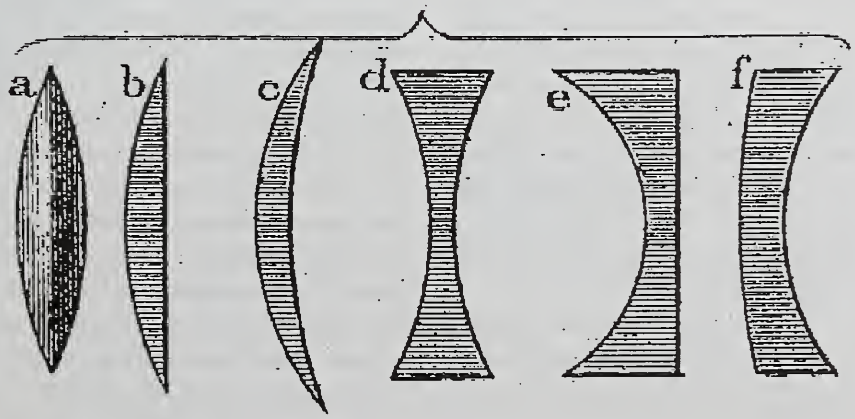
❖

A careful medical history and physical exam are the best screens for conditions that should be dealt with prior to surgery. These medical conditions may require specific laboratory tests in order to determine the safety of a patient for surgery. In the absence of any significant medical problems a limited number of tests are probably indicated: a) hemoglobin in individuals over sixty years of age, b) electrocardiogram in patients over 40 years of age, c) blood sugar if not done within one year.[2]

SURGICAL CONSIDERATIONS

Preoperatively, the length of the eye (measured by ultrasonic biometry) and the corneal curvature are used in a formula to determine the optimal power of the intraocular lens which is to be implanted. Depending on the specific situation, one may attempt to equal the refractive error of the fellow eye to achieve balance, aim for excellent distance acuity, or provide a myopic refractive error to allow reading without correction.

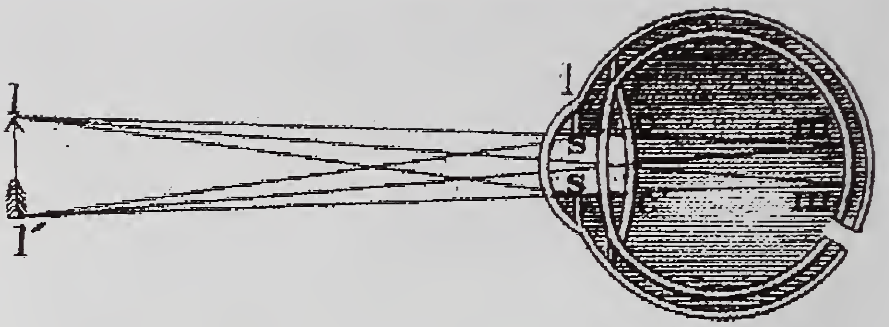
Although cataract surgery is performed with many variations, many aspects of the procedure are now universal. Leaming annually surveys American cataract surgeons to determine practice preferences and the percentages discussed below come from his most recent 1997 survey.[2] The anesthetic technique has been changing over the past few years with a decrease in the use of a local regional injected anesthetic from 99% in 1990 to 68% in 1997. The use of topical anesthesia alone or combined with intracameral unpreserved lidocaine has recently become more prevalent, increasing from 2% in 1993 to 30% in 1997. More than half of surgeons who perform more than 600 cases yearly employ this method of anesthesia administration. The avoidance of complications related to the use of a needle, such as orbital hemorrhage or inadvertent intravascular, intrathecal, or intraocular injections



have fostered this transformation. Topical anesthesia provides the patient with the ability to detect pain as an incipient indicator of choroidal hemorrhage, permitting exceptionally early surgical responses to this dreaded intraoperative complication. Patients generally prefer topical anesthetic for several reasons. For one, they are able to see immediately after surgery. Also, they do not need a postoperative patch and shield since normal ocular and lid motility is preserved with topical anesthesia. Lastly, patients dislike receiving an injection near the eye and the discomfort associated with it.

Traditionally cataract surgery has been performed through a superior scleral incision. This is still the most common site although the incidence has dropped from 75% in 1993 to 41% in 1997. This drop has been reflected in the dramatic increase in the use of temporal clear corneal or limbal incisions in 33% of cases. The impetus for this change has been the discovery that a temporal incision is more astigmatically neutral, provides better visualization during surgery, a better red reflex, easier manipulation of the phacoemulsifier handpiece, better drainage of fluid, and avoids the awkward approach to the eye caused by a prominent brow. Visual recovery is faster since there is a greater distance between the incision and the center of the visual axis than with a superior incision.

Ninety-seven percent of ophthalmologists now use phacoemulsification (employing a miniature ultrasonic fragmentation and aspiration device). (Figure 2) This device has allowed incision size to decrease, since only a 2 to 3.2mm incision is required for the removal of the lens by the phacoemulsifier tip. Much attention to incision design has occurred in the past few years and self-sealing incisions are now the norm, as evidenced by the fact that no-suture techniques have increased from a preference level of 1.2% in 1990 to 72% in 1997.

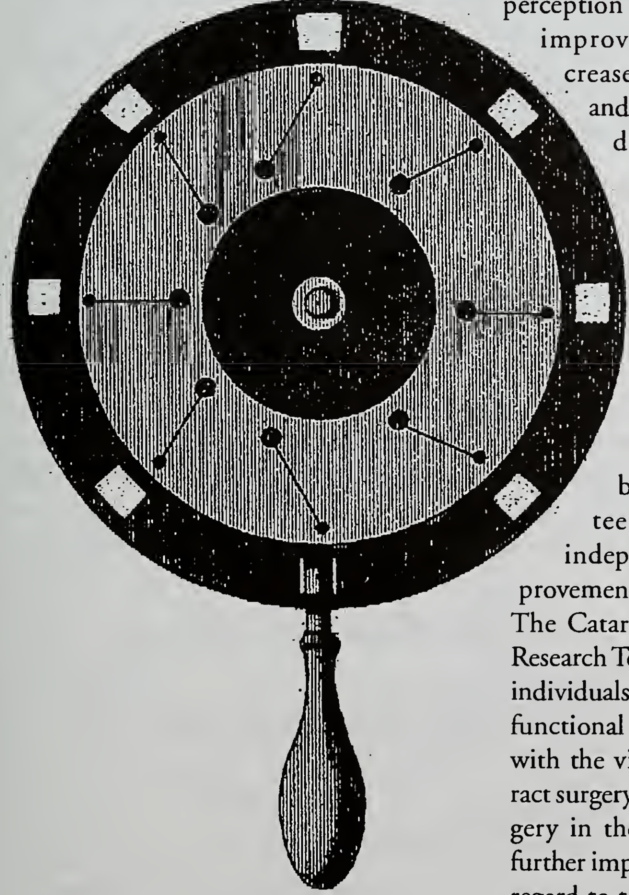


The changes in required incision size and the fact that smaller incisions can easily be made self-sealing has led to changes in the preferred intraocular lens materials. In 1986, in an era when surgery was performed through larger incisions, 98% of ophthalmologists preferred rigid polymethyl methacrylate (PMMA) lenses that require 5.25 to 7.5 millimeter incisions for implantation. In 1997 Leaming found that 60% of surgeons favor foldable lenses which can be implanted through 2 to 3.5mm incisions. The first foldable implants were made of silicone and this material is still preferred by 20% of ophthalmologists. Since their recent approval by the FDA, acrylic lenses have become the most popular foldable intraocular lenses, with a preference rate that has grown from 2% in 1994 to 38% in 1997. This shift has been driven by the lower incidence of posterior capsular opacification with acrylic lenses, the perceived better biocompatability of acrylic and hydroacrylic plastics when compared to silicone, and the slower and more controlled unfolding characteristics of these lenses.

A relatively new development has been the use of multiple posterior chamber lens implants in the same eye. Usually two lenses are used together. These "piggyback" lenses are used in several situations with excellent results. When a patient is very hyperopic, very strong intraocular lenses are required to correct vision. Lenses are usually only manufactured in only powers up to 30 diopters. Above this power, optical aberrations become a problem and interfere with visual quality. In the past, one would use a 30 diopter lens and leave the patient with residual postoperative hyperopia. Now, the proper power for correction can be divided between two lenses that are both implanted at the time of cataract surgery, one in front of the other, within the lens capsule. Not only do the two lenses provide a better quality image but they also provide greater depth of field. Some patients with piggyback lenses can even read when basically corrected for distance. Piggyback lenses have also helped patients with anisometropia (an imbalance of refractive error between the two eyes). In the past an intraocular lens exchange would have been considered. Explantation of the previously implanted lens was often fraught with complications due to adhesions. The determination of the correct power for the new lens to be implanted was not at all accurate since the initial imbalance could have been caused by one or more of many factors: a) the original lens power could have been incorrectly calculated, b) the lens power could have been mislabeled by the manufacturer, c) the lens could be further or closer from the cornea than usual. As a result, selecting the "proper" power of replacement lens based on the preoperative refractive error did not always yield correct results. By implanting a second "piggyback" lens in front of the preexisting incorrectly powered lens, one needs to correct only for the difference between the desired final correction and the current correction. This difference is the same regardless of the cause of the difference, and the correct additive intraocular lens strength can be easily and accurately determined.

In the past the goal of cataract surgery was to restore vision clouded by lens opacities. Today, in an era of concerns for quality of life, restoration of vision has been supplanted by a drive to optimize the patient's visual result. Cataract surgery has become a refractive procedure as well as a sight restorative one. Thus the ophthalmic surgeon attempts to correct any preexisting myopia or hyperopia by carefully determining the ideal intraocular lens power. Many ophthalmologists create a monovision system when both eyes require surgery, with the goal of having the patient's dominant eye corrected for distance and the other for near. Many patients do well with a mild imbalance and are able to see for both distance and reading without spectacles.

In 1997 the FDA approved a multifocal intraocular lens for implantation allowing for binocular uncorrected near and distance vision. This lens simultaneously creates a focused image at both distance and near. For patient satisfaction it must be implanted in both eyes and thus requires a patient with bilateral cataracts who wishes to commit to cataract surgery in both eyes within a short period of time since multifocal lenses must be implanted in both eyes in order to achieve patient satisfaction. There is some decrease in contrast sensitivity with multifocal lenses. In order to obtain a good outcome intraocular lens power calculation must be precise and astigmatism absent or surgically minimized.



Astigmatism (a condition where nonsphericity of the cornea induces image blurring) causes decreased postoperative acuity without spectacles. Astigmatic corneas are somewhat elliptical. By making precise, deep, linear or arcuate incisions in the peripheral cornea perpendicular to the steepest axis of curvature with a diamond knife, this axis is flattened and the cornea is rendered more spherical. Astigmatic keratotomy can now be performed just before beginning cataract surgery in many patients with significant astigmatism. A lens implant designed to correct astigmatism was released in November 1998.

Quality of Life Considerations

Improving patients' visual function through cataract surgery has been shown to have significant positive effects on their overall quality of life. The United States Department of Health and Human Services has determined that the benefits of improved visual function include such obvious factors as: improved reading ability, better uncorrected vision, improved depth perception and binocular vision, improved color vision, increased mobility (walking and driving), reduced glare disability, improved color vision, improved performance of the tasks of daily living, and the ability to continue or resume an occupation.[4] Less obvious changes include an increased sense of mental well-being, greater self-esteem, increased independence, and an improvement in social interaction. The Cataract Patient Outcomes Research Team found that 90% of individuals noted improvement in functional status and were happy with the visual outcome of cataract surgery in their first eye.[5] Surgery in the second eye leads to further improvement, especially in regard to tasks requiring binocularity, such as reading, driving, and general ambulation.

Cataract surgery has gone through many profound changes in recent years, including the indications for the surgery, the surgical techniques employed, and the quality of postoperative visual acuity. These factors have allowed modern cataract surgery to offer patients not only rehabilitation of their visual function, but also a transformation in their quality of life.

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In Office Retinal Detachment Surgery: Pneumatic Retinopexy, A Rhode Island Experience

Lory Snady-McCoy, MD

Pneumatic retinopexy (PR) is a new procedure that has dramatically changed how retinal surgeons manage retinal detachments. In contrast to standard scleral buckle surgery for retinal detachment repair, PR is performed in the office with local anesthesia and requires no anesthesia monitoring. It uses no incision, has low morbidity, low cost and good results.

Since the 1930's, the scleral buckling (SB) procedure has been the main procedure used for primary retinal detachment repair. When an encircling element (often made of silicone) is placed around the eye, vitreoretinal traction is relieved. In addition to surgically implanting this buckle, cryotherapy or laser is also performed to create a scar around the retinal break. Subretinal fluid is often drained externally. If there is extensive retinal fibrosis present, surgical removal of the vitreous (vitrectomy) can be performed in conjunction with SB. The success rate of SB is high, with 80-90% of retinas reattached.

Complications of SB include hemorrhage, infection, new retinal breaks, redetachment, glaucoma, diplopia, refractive error change and inflammation. In addition, visual acuity is reduced in the early postoperative period. An operating room and monitored anesthesia are needed.

Pneumatic retinopexy, a term coined in 1986 by Hilton and Grizzard,[1] was introduced to decrease some of the more severe complications of SB by avoiding the encircling element and the drainage of subretinal fluid. PR involves the injection of a gas bubble into the vitreous cavity of the patient's eye. The patient is then positioned so that the bubble "presses" against, or tamponades, the retinal break, thus closing it. When the retinal break is closed, the passage of vitreous fluid into the subretinal space is prevented and normal physiologic mechanisms then reabsorb the subretinal fluid. As in the standard SB, retinal cryotherapy or laser photocoagulation is performed to create a permanent chorioretinal adhesion. (Figures 1 and 2)

PR works well compared to SB in select cases. A controlled, prospective, randomized, collaborative study comparing PR and SB in 198 patients found that reattachment with one operation was 73% with PR vs. 82% with SB.[2,3]

This paper reviews 32 consecutive cases over 4 years in which pneumatic retinopexy was used to treat retinal detachment.

Abbreviations Used:

PR	pneumatic retinopexy
PVR	proliferative vitreoretinopathy
SB	scleral buckling

PATIENTS AND METHODS

Thirty-one patients (32 eyes) were identified as having undergone PR by the author. Their charts were reviewed and data collected regarding age, eye, best preoperative and postoperative vision, present vision, postoperative complications and procedures and past ocular history. (Table 1) Initially, the criteria used for consideration of PR were based on the indications originally described by Hilton and Grizzard. The retinal break or group of breaks involved no greater than one clock hour of the peripheral retina and were located within the superior 270 degrees of retina. Relative contraindications included extensive retinal fibrosis (proliferative vitreoretinopathy), the inability of the patient to maintain head positioning to ensure proper placement of the gas bubble against the retinal break, and mental incompetence. Although Hilton and Grizzard considered severe glaucoma a contraindication, several patients in this group had glaucoma and the procedure was performed without ill effect.

The procedure was performed as originally described with some modifications. Prior to surgery, each patient was given a peribulbar injection of anesthetic following topical cycloplegia, antibiotics and installation of a 50:50 solution of Betadine and balanced salt solution. Diamox 500mg Sequel was given orally to lower the potential risk

Table 1. Preoperative Ocular History in Eyes Treated with Pneumatic Retinopexy

Cataract surgery	12	Macular pucker	2
Peripheral retinal lattice degeneration	10	Dislocated lens during cataract surgery	1
Vitrectomy surgery	6	Trauma	1
Glaucoma	5	Uveitis	1
Proliferative diabetic retinopathy	3	Macular hole	1
Scleral buckle surgery	2	Proliferative vitreoretinopathy	1

Table 2. Preoperative and Postoperative Vision in Eyes Treated with Pneumatic Retinopexy

Vision	Preoperative	Postoperative
20/20-20/25	9	22
20/30-20/40	10	4
20/50-20/70	3	1
20/80-20/100	2	1
20/200-20/400	6	3
CF - HM	2	1
Total	32	32

(CF=Count Fingers, HM=Hand Motion)

of a postoperative rise in ocular pressure unless the patient had a history of sulfa allergy. The retinal break or breaks were treated with cryotherapy. A 30-gauge needle was used to drain a small amount of aqueous humor from the anterior chamber under sterile conditions. Then another 30-gauge needle was used to inject 0.3-0.5 cc's of sulfahexafluoride (SF6) gas into the vitreous cavity. SF6 was used for several reasons: 1) it is relatively nontoxic; 2) it expands to 2.5 times its initial volume to aid in tamponading the retinal break; and 3) it has low solubility and remains in the vitreous cavity for 7-10 days before being resorbed. After the central retinal artery was checked for possible occlusion and the position of the gas bubble was confirmed with indirect ophthalmoscopy, antibiotic ointment was instilled and the eye was patched. An arrow was drawn on the pad toward the clock hour of the retinal break to help the patient keep correct head positioning until the next day's examination. The procedure usually took 30 minutes. Another Diamox Sequel was given to the patient to take 6 hours after the procedure, if possible, when the gas bubble would reach its greatest expanded volume.

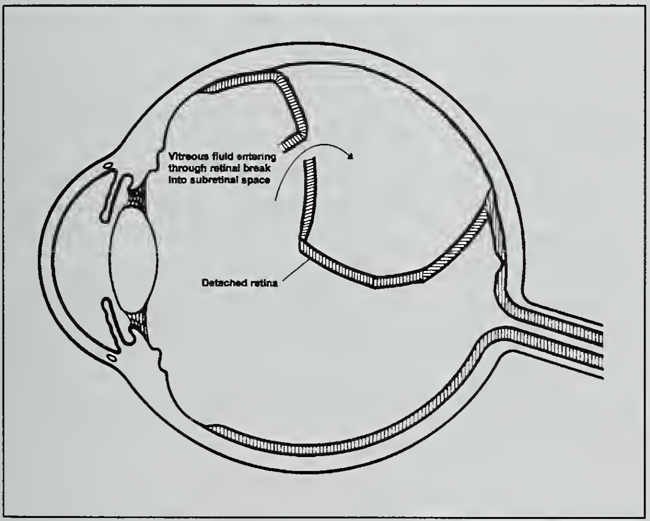
Vitreous fluid entering through retinal break into subretinal space
Detached retina

Figure 1. Retinal detachment.

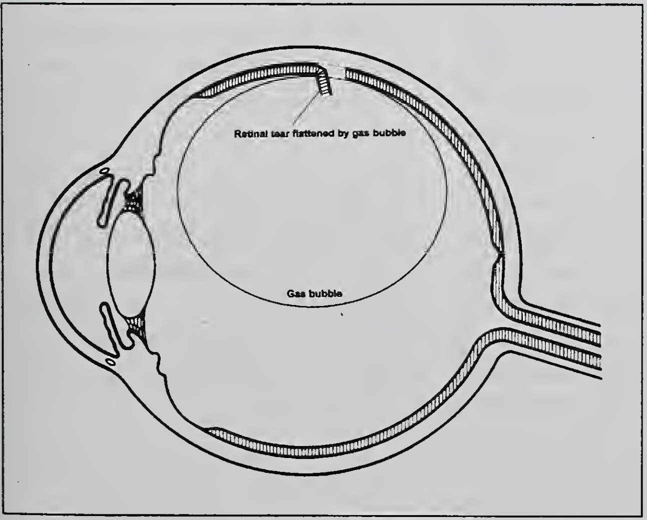

Figure 2. Retinal detachment treated by pneumatic retinopexy.

Following examination the next day, the eye patch was discontinued and a topical antibiotic-steroid solution was used 3-4 times daily for one week. All patients were instructed to keep their heads positioned correctly as much as possible during the day. Because of the potential of cataract formation from SF_6, they were told to lie on their sides at night, being careful not to lie on their backs. The patients were re-evaluated again in 3-5 days. They generally returned to work 2-4 weeks after surgery.

RESULTS

Thirty-two eyes in thirty-one patients underwent PR by the author between October, 1994, and October, 1998. Fourteen patients were male, 17 were female. Age range was 26-80 years old. The distribution of eyes was equal (16 right, 16 left eye). One patient underwent PR in both eyes. Range of follow-up was 1 month to 4 years, 1 month. Thirty eyes had been followed for greater than 6 months. The original criteria for PR (retinal break of breaks no larger than one clock hour within the 270 degrees of superior retina) were expanded to include patients with larger breaks (2 cases) and multiple breaks over a larger area (4 cases). The contraindications (proliferative vitreoretinopathy [PVR]), inability to maintain positioning, mental incompetence and severe glaucoma) were not strictly followed. One case of previous uveitis and PVR chose to undergo successful PR because she was 6 1/2 months pregnant. Five eyes had a history of glaucoma. One patient had undergone glaucoma surgery to her fellow eye one day prior and chose PR for better immediate postoperative vision in the eye with retinal detachment while the fellow eye healed.

Fourteen eyes had undergone some type of procedure prior to PR, including phacoemulsification cataract surgery with artificial intraocular lens placement (11 cases), some of which were complicated by a ruptured lens capsule (4 cases). One case was aphakic (without any lens) due to trauma as a child. Six cases had undergone vitrectomy surgery and two cases had undergone recent SB retinal detachment repair. Ten eyes had peripheral retinal lattice degeneration, a condition that predisposes to retinal breaks and detachment. Despite these complicated pre-operative situations, nineteen eyes had successful reattachment of the retina with only one procedure. Sixty-three percent were reattached if one case that underwent only PR and supplemental laser for a new break found the first postoperative day is included. A subgroup of 9 cases who had not undergone previous cataract surgery and had a localized detachment with only one break had a 100% one-procedure success rate.

Four cases required repeat PR. Two cases developed a new retinal break without detachment, both treated successfully with laser. Four cases redetached and required SB with vitrectomy. Two cases developed post-operative preretinal macular fibrosis (macular pucker) requiring vitrectomy.

Of the 13 cases requiring an additional procedure, 92% were performed within 2 months of the PR. More cases required additional surgery in patients who had undergone previous cataract surgery (58%) than not (35%). The four eyes that redetached and required a hospital based procedure underwent this procedure at 1,2,8 and 11 months following PR. Three of four eyes that required SB/vitrectomy had significant past ocular disease including one status post vitrectomy for dislocated lens nucleus during cataract surgery and two with extensive 360 degrees of peripheral retinal lattice degeneration. In these three cases, the macula, the central most part of the retinal responsible for 20/20 vision, remained attached. The fourth patient ignored symptoms of decreased vision for over one week and presented with an almost total redetachment due to a new tear. He later underwent second vitrectomy for macular pucker. Despite this, vision ultimately improved to 20/30+2.

Visual results in the series were good. (Table 2) Preoperatively, nineteen of 32 patients (59%) had 20/40 vision or better (legal driving vision). Postoperatively, 26 of 32 patients (81%) had 20/40 or better vision. Of the six patients who did not achieve 20/40 vision, five had poor vision due to underlying retinal disease. All but one patient had equal or better vision following PR. Reports have shown that final vision after PR is better than SB in patients whose retinal detachment involved the central macula. Ten cases presented here had retinal detachments involving the macula, 80% with 20/100 or worse vision preoperatively. Following surgery, 90% had 20/50 or better vision.

Given the lower morbidity, expense and complication rate, it seems reasonable to attempt PR in select patients.

CONCLUSIONS

This paper reviews 32 consecutive cases over 4 years in which pneumatic retinopexy was used to treat retinal detachment. Despite the inclusion of complicated retinal detachments, results were favorable: 59% of patients required only this one procedure and 81% had 20/40 or better postoperative vision. Most cases were successfully treated with only in-office procedures. This compares favorably with other reported cases [4].

In addition to technical success, the cost of the surgery was also good compared to SB. The total expense of PR including reoperations was estimated to be 60-70% of the cost of the same cases if they were treated with SB in a hospital setting, even assuming an unrealistic 100% success rate of the SB procedure. Moreover, the patients' subjective impressions regarding PR vs. SB favored PR. The four patients who underwent both PR and later SB/vitrectomy clearly favored PR even if they had undergone other postoperative procedures before SB/vitrectomy.

Lastly, although the overall one-procedure success rate of PR is not as high as SB in reattaching the retina, it does not lower the final anatomical or visual success rate if subsequent procedures are needed. Indeed, the final success rate of retinal reattachment was 100% in the cases presented here.

Given the lower morbidity, expense and complication rate, it seems reasonable to attempt PR in select patients. With further refinements of the procedure already suggested in recent literature,[4] it is probable that this lower-cost office-based surgery will be a viable option for many more retinal detachment patients in the future.

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New Perspectives in Corneal Transplant

Elliot M.Perlman, MD

The first person on record to suggest corneal replacement was de Quengsy. At the time of the French Revolution he had the idea of making an artificial cornea, using a glass similar to a watch crystal, framed with a silver rim (see cover).

He gave details on technique, counseling that a beautiful day should be chosen for the operation and that the patient should be lying flat on his back for eight days lest the implant fall out! We don't know whether the procedure worked: there is no record of anybody attempting it.[1]

Even though corneal transplant, however fanciful, had been conceived by deQuengsy (at least in principle), the first actual human transplant did not take place until 1906. For the next fifty years, transplants were rarely done. But with the establishment of eye banks in the 1950s, they became practical.

And when corneal storage medium was developed in the 1970s, corneal transplants became much more convenient — and successful. Today, corneal transplantation is the most successful tissue transplantation procedure in modern surgery, with a general success rate of over 90%. Last year 46,000 were performed in the United States (124 in Rhode Island).

In this article I will discuss current techniques involved in this procedure, including transplant indications, donor selection, tissue storage and matching, eye banking, corneal preservation, preoperative medical preparation and postoperative management. In addition, I will also briefly discuss other innovative forms of ocular tissue transplantation such as limbal grafting and amniotic tissue transplants.

INDICATIONS FOR CORNEAL TRANSPLANTATION

The normal cornea is a clear almost spherical dome. In addition to allowing light to enter the eye, the cornea bends (refracts) the incoming light to focus the image on the retina. In fact, the cornea does almost 70% of the refraction, while the lens of the eye does 30%.

Abbreviations Used:

HIV:	human immunodeficiency virus
ATP:	adenosine triphosphate
TBI:	Tissue Banks International
TDIC:	Tissue Distribution and Information Center
CCTS:	Collaborative Corneal Transplantation Study
HLA antigen:	human leukocyte-associated antigen

The normal cornea maintains its compact clear status because it is dehydrated compared to other body tissue (72% water vs. 98% water). This dehydration is maintained by the innermost layer of the cornea, the corneal endothelium (Figure 1). The endothelium has an ATP-dependent pump involving the transport of bicarbonate ion. Water leaves the corneal stroma following the resultant osmotic gradient.

Any disease that destroys the corneal endothelium can cause the cornea to hydrate and become cloudy. Since the endothelial cells cannot reproduce, the condition is often irreversible. The only way to resupply endothelial cells is by corneal transplantation.

The most common cause of corneal endothelial cell loss and subsequent corneal hydration is damage from intraocular surgery such as might occur after cataract operations. Another relatively common hereditary disease that causes endothelial cell loss is Fuchs' corneal dystrophy. Both these conditions are managed by corneal transplantation.

In order to provide good vision, the cornea, in addition to being compact, must be perfectly clear. Conditions such as corneal lacerations and corneal scars from infections can lead to corneal opacities in the stroma that preclude good vision. Again, corneal transplantation works well in these conditions to restore sight.

Lastly, the cornea must have a nearly spherical shape to refract light properly. A relatively frequent cause of poor vision from corneal curvature problems is keratoconus. In this condition, the cornea becomes cone-shaped, thereby greatly distorting the retinal light image. In the early stages of keratoconus, glasses or rigid contact lenses are often all that is needed for good vision. However, if these modalities fail, corneal transplantation is usually successful in restoring the appropriate shape of the cornea.

DONOR SELECTION

Corneal tissue from almost any age donor is suitable for corneal transplantation. Tissue from very young donors (less than 2 years old) is often very steep and flexible and is generally not used. There is no absolute upper age limit, but tissue from donors over 75 years old is generally not used. To ensure survival of the corneal endothelial cells, the time between death and enucleation should be less than eight hours.

Using detailed donor history and blood screening techniques, eye banks make every attempt to avoid transmission of diseases from the donor to the host. Such efforts focus on microbial infections (especially bacterial and viral diseases) but also deal with other possible transmissible diseases such as malignancies.

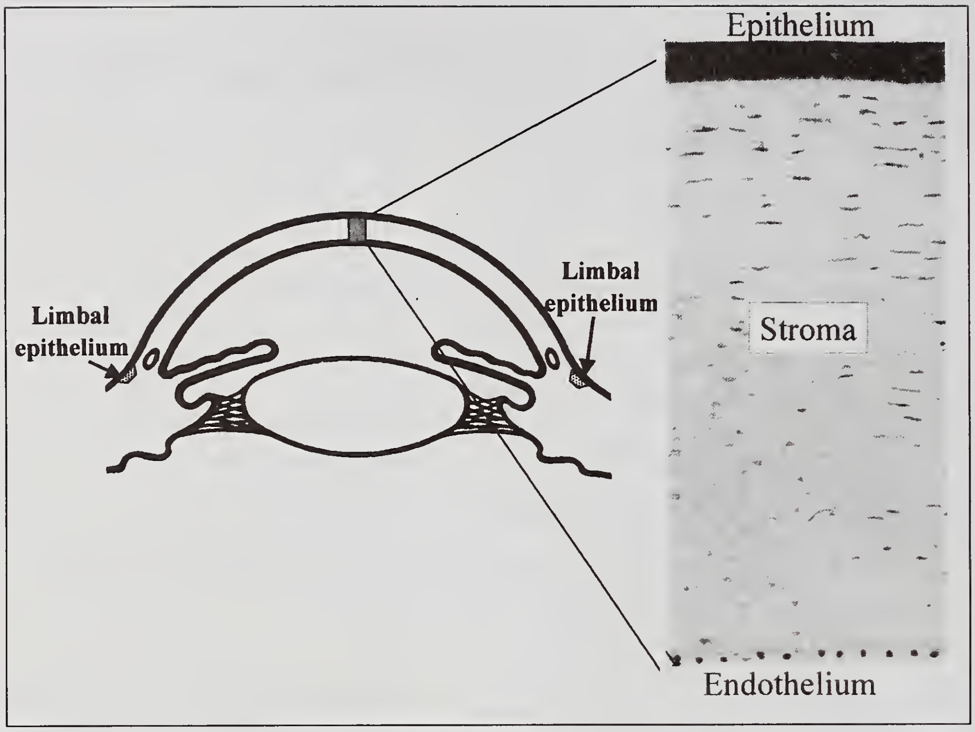

Figure 1. Corneal anatomy

BACTERIAL TRANSMISSION.

In addition to excluding all donors with sepsis from any cause, eyebanks add antibiotic to all corneal preservation solutions.

VIRAL TRANSMISSION.

Because of a case of possible transmission of hepatitis B virus from an infected donor in 1986, the Eye Bank Association of America requires a screening test for hepatitis B surface antigen. Donors also must have a negative screening for hepatitis C virus, although there are no known cases of transmission of this disease.

There are no known cases of HIV transmission from corneal donors, despite a report of corneal transplantation performed inadvertently using tissue from two HIV-infected donors. Nonetheless, HIV, HIV-1 and HIV-2 antibody tests must be negative before tissue is released for transplantation. Because HIV antibodies may not be detectable in 3-6% of cadavers with AIDS and because of the latency period for positive antibody response, persons at high risk for HIV infection are excluded from corneal donation.

Both rabies and Creutzfeldt-Jakob diseases have been transmitted by corneal transplantation. For this reason, these diseases and all potential donors with neurologic diseases of unknown etiology are excluded from donation.

Transmission of malignancies.

There has been one reported case of retinoblastoma transmitted by corneal transplantation, but no reports of transmission of choroidal melanoma. The latter cases may be suitable for donor tissue. There has been no reported transmission of systemic malignancies by corneal transplant. Nonetheless, tissue from patients with leukemia or disseminated lymphoma is not used.

TISSUE STORAGE MEDIUM

Until the mid-1970s, donor corneal tissue was stored as a whole eye in a moist chamber. Tissue stored in this fashion will survive only 24-48 hours. Part of the problem is that decomposition of the rest of the ocular tissue is toxic to the corneal endothelium. Current preservation technique therefore involves surgically removing the corneoscleral rim from the rest of the globe before storage and placing only this tissue in the preservation medium. Tissue prepared in this fashion resembles a soft contact lens in appearance and texture (Figure 2).

The donor corneoscleral rim is placed in a bottle of preservation medium. The donor endothelium can actually be examined while it is in the bottle, using a special specular microscope. In this fashion, a fair assessment of tissue viability can be made prior to transplantation.

If the tissue is satisfactory, the bottle is placed in a styrofoam container surrounded by ice for shipping. (Until the time of surgery, the bottle can be removed from the container and stored in an ordinary refrigerator at 4 degrees C.)

Most eye banks in the United States use Optisol GS storage medium. This consists of a buffered tissue culture medium, 2.5% chondroitin sulfate, 1% dextran 40, gentamicin and streptomycin (the "GS"). Also added to this solution are a proprietary mixture of sodium pyruvate, nonessential amino acids, antioxidants, ATP precursors, ascorbic acid and vitamin B_{12}. Corneal tissue stored in Optisol GS may last up to two weeks; however, I avoid using tissue stored longer than a week.

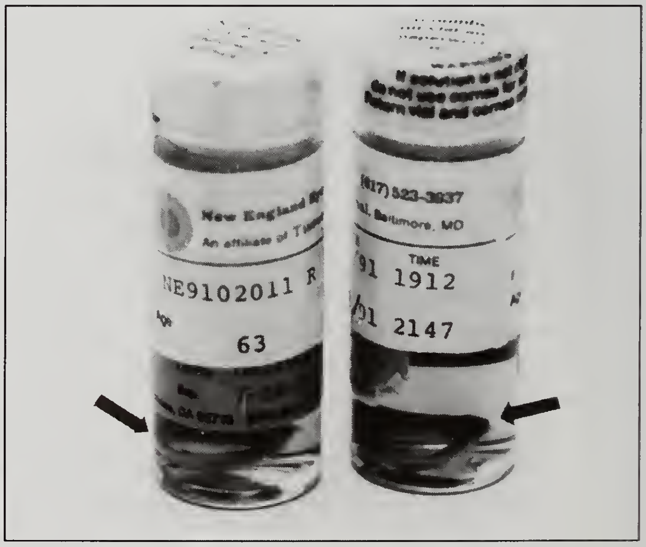

Figure 2. Tissue in storage medium

TISSUE MATCHING

From knowledge of other types of tissue transplantation, one would logically assume that tissue matching of donor with recipient would make corneal transplant rejection less likely. Because of conflicting results from many earlier studies addressing this issue, ophthalmologists at a number of centers in the United States conducted a prospective, randomized controlled clinical trial and published their results in 1992.

The paper, called the Collaborative Corneal Transplantation Study (CCTS)[2], examined the question of whether HLA-A, HLA-B, HLA-DR or ABO compatibility affects the outcome of high-risk keratoplasty patients. The results showed that HLA-A, HLA-B or HLA-DR matching had no effect on overall transplant survival or incidence of rejection. The CCTS further found that ABO blood group matching had a favorable effect on transplant outcome, although the reasons are unclear.

Using this information, eye banks could theoretically match high-risk transplant patients fairly easily with ABO compatible donors, although this is rarely done. In fact, 70% of donor and recipient pairs would be ABO compatible by chance alone.

EYE BANKING SYSTEM

One of the major advances in corneal transplantation today involves the unification of tissue banks on a national scale through Tissue Banks International (TBI), a consortium of 26 eye banks coast-to-coast. Founded in 1962, it is a non-profit, non-governmental organization and is the largest non-profit eye and tissue-banking network in the world.

The New England Eye Bank in Boston, an affiliate of TBI, provided 2354 corneas (1184 donors) for transplantation in 1997. Rhode Island donors accounted for 100 of these corneas.

TBI's Tissue Distribution and Information Center (TDIC) is a computerized system which quickly and efficiently enables surgeons throughout the nation to obtain quality eye tissue for their patients. Local tissue from Rhode Island is first distributed through New England Eye Bank, then regionally, then nationally, so that it does not go to waste. If insufficient tissue has been recovered locally on a given day to meet local surgeons' needs, TDIC will make eye tissue available from another TBI eye bank. TBI has thus virtually eliminated waiting lists for corneal transplant surgery in locations serviced by member eye banks.

The donor tissue is transported free as a courtesy of bus companies or commercial air carriers. (The tissue is kept in the plane's crew cabin, not with the luggage or other packages.)

Corneal transplantation is the most successful tissue transplantation procedure in modern surgery, with a general success rate of over 90%.

PATIENT PREPARATION AND OPERATIVE TECHNIQUE

Because of the availability of corneal tissue afforded by the eye bank consortium and modern corneal storage medium, corneal transplants can be scheduled in advance with as little as several days' notice, much like routine outpatient cataract surgery. We no longer use a waiting list as we did even a decade ago, where patients waited months, or even years, for donor tissue. In my experience, it is uncommon (perhaps 1-2% of all cases) to have to cancel a transplant because tissue is unavailable.

Since the cornea is an avascular structure, the risk for serious bleeding problems intraoperatively is very small. Consequently, most corneal surgeons do not stop their patients' oral anticoagulants (eg aspirin or Coumadin) prior to surgery.

Almost all corneal transplants today are done using local (regional) anesthetic, most commonly peribulbar injections (injections around the eye), with the patient awake receiving some intravenous sedation as needed.

The corneal transplant procedure itself takes about an hour. The donor tissue - similar to a contact lens in appearance - is placed epithelial-side down, usually in a small Teflon bowl-shaped block. A trephine is used to punch out the donor disk. A similar trephine is used on the patient's cornea. A typical transplant uses 8-mm diameter disks. The donor is sutured into position using either interrupted or continuous sutures of 10-0 monofilament Nylon (about 22 microns in diameter) (Figure 3). All surgery is done using an operating microscope.

At the end of the surgery the eye is patched and the patient is allowed to go home. The patch is removed on the first postoperative day and left off. I generally require little to no limitation of the patient's activities except that I advise the patient to wear protective glasses in the daytime and a plastic protective shield while sleeping. Postoperative medications usually are topical corticosteroids and antibiotics.

In the early postoperative period (the first 4-6 months), the corneal graft contour is not usually spherical, so the patient's vision is not very good. Much of this irregularity is due to unequal suture tensions. Therefore, during this time, the corneal surgeon removes sutures or adjusts suture tension to regularize the corneal contour. To aid this process, most corneal surgeons make use of modern corneal topographers, computerized devices that produce corneal contour maps and indicate which sutures should be removed or adjusted. Typically after 4-6 months, the patient achieves the anticipated visual result.

LIMBAL GRAFTS AND AMNIOTIC TISSUE GRAFTS

Good vision relies not only on the shape and clarity of the corneal stroma but also on the integrity and smoothness of the corneal epithelium - the cells which line the outer surface of the cornea (Figure 1). These cells are normally constantly replaced in response to minor injuries such as corneal abrasions.

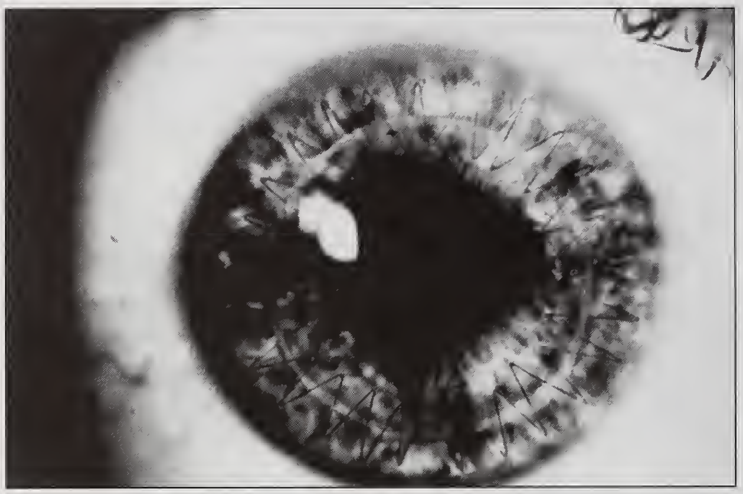

Figure 3. Postoperative appearance of Corneal Transplant

The source for the corneal epithelial cells is a deep layer of epithelial cells located in the corneal limbus - the transition zone between the cornea and the sclera. If the limbal area is severely damaged - as might occur in an alkali burn - the corneal epithelium cannot regenerate properly, leaving large areas of exposed corneal stroma. These areas of epithelial defects can lead to serious infections of the underlying corneal stroma as well as proteolytic destruction ("melting") of the stroma.

A typical corneal transplant transfers the central 8mm of the 11mm cornea and therefore does not transfer limbal tissue. Consequently, in recent years we have begun performing limbal grafts in order to transplant limbal epithelial cells. The tissue is obtained either from a healthy contralateral eye - as in the case of unilateral alkali injury - or from donor tissue obtained from a relative of the patient or from a cadaver. These limbal grafts have allowed visual restoration in eyes formerly considered untreatable.

Another unusual type of transplanted tissue involves the use of amniotic membrane grafts. These grafts may be helpful in situations where the corneal epithelium is healing poorly - such as in eyes in which the sensory nerve is damaged (neurotrophic keratitis).

Amniotic membrane (amnion) is the innermost layer of the placenta. It is obtained from human placentas at the time of elective cesarean section. The tissue is frozen onto a piece of nitrocellulose filter and shipped in dry ice. At the time of surgery, the amniotic membrane is thawed, cut to the appropriate size, and sutured over the defect in the corneal epithelium.

In addition to acting like a bandage over the defect, the membrane may also suppress scar tissue formation and exclude inflammatory cells. Since the membrane is acellular, it does not provoke an immunological response.

CONCLUSIONS

In the past twenty years, corneal transplants have gone from rarely performed procedures with a poor success rate to routinely scheduled surgeries with a very high success rate. We can hope that our progress in the next millennium will be equally impressive. For instance, we still await a successful method of cryopreservation in which corneal tissue could be preserved indefinitely. In addition, we might soon be able to transplant cultured corneal endothelial cells for endothelial diseases instead of requiring the entire cornea to be grafted.

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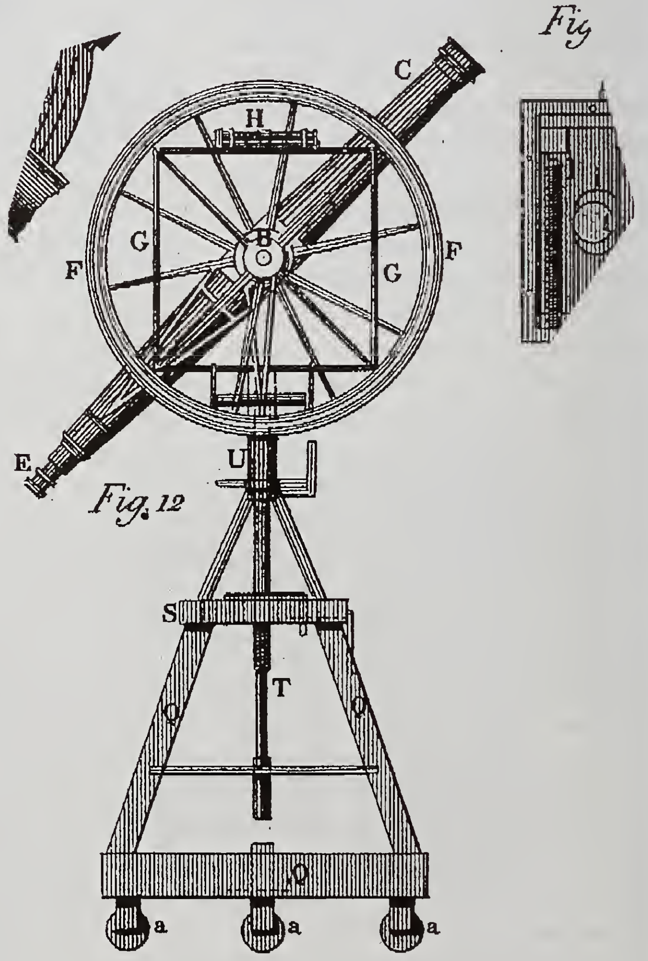

Systemic Autoimmune Diseases and Their Effect on the Eye

Thanos Zervos, MD, and William Tsiaras, MD

Inflammation is a complex process of tissue reaction set in motion by one or more antigenic stimuli. These stimuli cause changes in vascular permeability with inflammatory cell migration into organs or connective tissue causing varying degrees of tissue destruction and ultimately scarring. Most of these processes are mediated by cytokines. Cytokines are molecular messages exchanged between cells that control the myriad responses of cells. Advances in molecular medicine are shedding new light on many of these complex cellular processes. In no other group of diseases has there been more activity in this new area than the diseases which affect the immune system.

In some of the systemic immune diseases there is a misdirected attack against ocular tissues. In this paper we present a few of these diseases and discuss some of the current thinking about disease pathogenesis, diagnosis and treatment.

SJOGREN'S SYNDROME

Primary Sjogren's syndrome (SS) is a systemic autoimmune disease that is characterized by keratoconjunctivitis sicca and xerostomia resulting from lymphocytic infiltrates of the lacrimal and salivary glands. There is a debate regarding the diagnostic criteria for SS. Much of the debate centers around the need for positive salivary gland biopsy as an important criterion for diagnosis. Non-invasive studies, such as salivary gland scintinography, may, in association with other clinical findings, be adequate for establishing a diagnosis. Currently research groups are attempting to develop an acceptable diagnostic system for SS.

Patients with SS have immunologic abnormalities including impaired lymphocytic function and lymphoreticular neoplasms. Further, certain HLA and non-HLA genes appear to predispose susceptible individuals to develop this disease. Non-HLA genes include the Y-chromosome-linked autoimmune accelerator and the gene for protein tyrosine phosphatase. It has also been shown that non-HLA genes influence the age-related decrease in salivary and lacrimal gland function. Other important components in the pathogenesis of SS include factors that allow the activation of autoimmune T cells of the thymic system. These cells suppress the down-regulation of the ongoing autoimmune reaction by mechanisms of apoptosis.[1]

Recent publications have addressed important clinical observations such as the relationship between SS and Chronic Fatigue Syndrome (CFS) or fibromyalgia (FM), multiple sclerosis-like central system involvement and the potential association between SS and silicone breast implants. The relationship between SS and CFS or FM is currently controversial. There is evidence that sicca symptoms are much more common in CFS and FM. Several investigators believe that these symptoms are a result of altered autonomic function at the level of locus ceruleus in the hypothalamus. Therefore it is important for the rheumatologist to distinguish symptoms due to CFS or FM from those resulting from an autoimmune process, such as SS, in order to decide on a the treatment plan.

Although features of multiple sclerosis have been reported in patients with sicca symptoms, it is now clear the SS patients can have demyelinating lesions of the central nervous system. The frequency of this association is controversial. Several retrospective uncontrolled studies have demonstrated an increased prevalence of sicca symptoms in patients with silicone breast implants. The Food and Drug Administration recently reviewed the literature and stressed the hazards of relying on anecdotal case reports only for silicone breast implants.[1]

Abbreviations Used:

AP-1	activator protein-1
SS	Sjogren's syndrome
HLA	human leukocyte-associated antigen
CFS	chronic fatigue syndrome
FM	fibromyalgia
TAO	thyroid associated ophthalmopathy
IgA	immunoglobulin A
TON	thyroid optic neuropathy
ANA	antinuclear antibody
NSAID	nonsteroidal anti-inflammatory drugs
PUK	peripheral ulcerative keratitis
RA	rheumatoid arthritis
CVD	collagen vascular disease
WG	Wegener's granulomatosis

Several studies addressing new topical and systemic treatment protocols have been published over the last few years. Based on canine dry-eye management techniques used by veterinarians for years, Gynduz and Ozdemir conducted a randomized double-blind, placebo-controlled trial using topical cyclosporin 2% in olive oil. Their results, after a 2-month follow-up time,[2] showed a statistically significant improvement in two objective measurements of tear function in patients with secondary SS who received topical cyclosporin. However, another common tear production test, the Shirmer's test, remained unchanged in these patients. In animal models of SS, Sullivan et al[3] found a beneficial effect for topical androgen therapy on the lacrimal gland and conjunctival surface pathology.

Thyroid Associated Ophthalmopathy (TAO)

Thyroid ophthalmopathy is the most common unilateral or bilateral orbital disorder in adults. It affects patients with active or treated Grave's disease, usually within one year of the manifestation of the hyperthyroidism. Thyroid ophthalmopathy can present in euthyroid or even in hypothyroid patients. The clinical presentation includes lid retraction, proptosis, restrictive myopathy (the inferior rectus and medial rectus are most frequently involved muscles), conjunctival congestion, exposure keratopathy, glaucoma, strabismus, and optic neuropathy.[4] Lacrimal gland inflammation is also frequently present. Recent studies have documented the presence of increased levels of manganese superoxide dismutase in the tears of patients with TAO and have also detected IgA class autoantibodies which react with fibroblasts in the orbit and skin. These findings support a central role for lacrimal gland inflammation in this process.[5]

There is now general agreement that the eye muscle and the orbital connective tissue compartments are both targets of the autoimmune reaction of TAO. Both humoral and cell-mediated mechanisms have been invoked. Histochemical studies show that the ocular muscles contain heat shock proteins and major histocompatibility antigens and can function as antigen-processing cells. The orbital fibroblast is known to be stimulated by a variety of soluble proteins including cytokines and adhesion molecules released in the context of an autoimmune reaction in the retroorbital connective tissue and fat. The orbital fibroblasts can subsequently produce excess glycosaminoglycans or directly damage the orbital tissues. There is also some evidence that the extrathyroidal abnormalities of Graves' disease may reflect a more generalized inflammatory disorder of striated muscle and connective tissue. Autoantibodies identified in the serum of patients with TAO react with orbital tissue proteins, as well as with proteins in similar tissues elsewhere in the body. The influence (positive or negative) of radioactive iodine treatment on the progression or the development of the ophthalmopathy remains controversial. Many groups are investigating the role of immunoreactivity against an abnormal or variant TSH-receptor protein in orbital tissues.[5]

The new challenge for a rational therapy in autoimmune-induced ocular inflammation is the clarification of the specific molecules causing disease.

◆

Thyroid optic neuropathy (TON) is seen in 5% of patients with thyroid ophthalmopathy and constitutes the most common cause of vision loss in these patients. Often patients with advanced optic neuropathy are free of symptoms and have a normal Snellen visual acuity. In order to prevent visual loss it is therefore important to periodically evaluate these patients with careful clinical examination, including evaluation of pupillary reaction, color vision and automated or Goldman visual fields. The most common visual field defects are central scotomas and arcuate or altitudinal (especially inferior) scotomas. The underlying process is thought to be either optic nerve compression from the inflamed extraocular muscles at the orbital apex or optic nerve ischemia.[4] The use of new diagnostic procedures, such as a radiolabeled somatostatin analog scan of the orbit and measurement of serum orbital tissue-reactive autoantibodies, may provide for a more efficient management of the thyroid optic neuropathy.[4]

Current treatment options include one or more of the following: systemic corticosteroids, orbital irradiation, surgical decompression of the orbit and possibly systemic immunosuppressive medications. Intravenous immunoglobulin has also been used with moderate success by several groups. The main disadvantage of this form of therapy is its expense and the possibility of transmitting viral infection, including Hepatitis B and C.[5] Oral corticosteroids in doses of 60-100 mg/day are the most frequently used medications for the treatment of TON. Intravenous methylprednisolone can also be used. Steroids can successfully control the conjunctival chemosis and eyelid edema but have little effect on proptosis, strabismus or chronic orbital fibrosis. The response to treatment is usually good, with patients achieving visual acuity of 20/40 or better in 55-60% of all cases. Initial responses in visual acuity and visual field should be evident within days, and maximum responses should occur within 6-8 weeks. Because of the high dosages and the prolonged duration of corticosteroids required, prevention and treatment of side effects is of great importance. Potential side effects include diabetes mellitus, gastrointestinal ulcers, bleeding, weight gain, hypertension, fractures, aseptic necrosis of the femoral head and psychosis. Prolonged treatment with high-dose steroids as the sole therapeutic modality beyond 8 weeks without response, or beyond 12 weeks even with improvement, is not recommended.

Patients whose optic neuropathy initially responds to steroids but later recurs during tapering, or patients in whom corticosteroids are contraindicated, should be considered for radiation therapy. Radiotherapy for thyroid orbitopathy was first used 50 years ago and became more prominent in the early 70ís. Current protocols include a low dose orbital irradiation from a 4.0-6.0 meV linear accelerator with blocking shields to minimize exposure to the lens and the retina. The usual dose is 2,000 cGy in 10 courses over 2 weeks. Side effects are minimal and most often involve a transient increase in orbital inflammation lasting usually 1-2 weeks. This treatment will reverse TON in 66-75% of patients. Several studies have shown a comparable efficacy of either oral corticosteroids or orbital irradiation for the treatment of thyroid ophthalmopathy; their combination, though, appears more effective than either modality alone.

Several studies have documented the effectiveness of orbital surgical decompression in treating thyroid orbitopathy. Through a transorbital, transantral or a transfrontal approach, decompression of the medial and inferior orbital wall is performed. Orbital decompression is probably the most effective treatment of TON, but has potentially serious side effects. Good candidates include: patients with rapidly declining vision or visual field who show no response to steroids; patients with TON and no obvious orbital inflammation; patients with prominent proptosis, or exposure keratopathy; patients with relative contraindications to radiotherapy, such as keratoconjunctivitis sicca, diabetic retinopathy, hypertension; or patients receiving chemotherapy. A complete resolution of visual field defects can be achieved in 44-54% with these approaches, whereas a partial improvement has been documented in additional 33-46%. Visual acuity can be improved in up to 82% in eyes with TON despite prior failure with corticosteroids or orbital irradiation. Median time until stabilization of visual acuity after decompression is approximately 2-3 weeks. Techniques with three or four wall decompression have also been reported with comparable results. Most patients with TON undergoing orbital decompression no longer require corticosteroids or other additional therapy. Surgical complications include diplopia, mainly seen in the transantral approach (25-64%, depending on the study), cerebrospinal fluid leak, lower lid entropion, intraoperative blood loss requiring transfusion, upper lip numbness, sinusitis, nasolacrimal duct obstruction and oroantral fistula.[4]

COLLAGEN VASCULAR DISEASE AND PERIPHERAL ULCERATIVE KERATITIS

Peripheral ulcerative keratitis (PUK—Figure) can be seen in a variety of infectious and inflammatory conditions, autoimmune or not, with a local or systemic spectrum of manifestations. Collagen vascular diseases (CVD) constitute a major etiologic factor, up to 50% of all cases in one study, with rheumatoid arthritis (RA) being the most common cause among CVD's (34% in the same study).

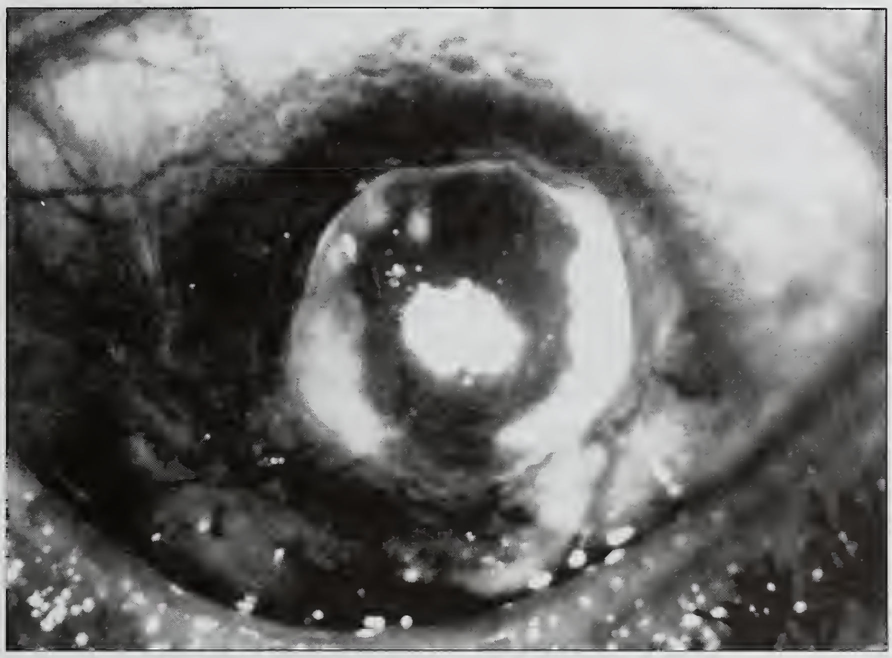

Figure. Peripheral ulcerative keratitis in patient with Wegener's granulomatosis.

The presenting ocular signs and symptoms include decreased vision (usually a result of an irregular, induced astigmatism), irritation, redness and pain. There is associated conjunctival scleral and episcleral inflammation, with episcleritis or scleritis. A peripheral, sterile corneal stromal infiltrate, which represents an accumulation of inflammatory cells, is usually seen. PUK and necrotizing scleritis have been associated with increased risk of death in patients with RA. The presence of PUK or necrotizing scleritis in RA patients is an indication that the disease has changed from a synovial microvasculitis to a systemic vasculitis. An increased number of PUK cases in RA patients undergoing eye surgery has been noted. It is believed that surgical trauma initiates an immune-complex mediated vasculitis in these patients. The PUK is frequently bilateral in RA patients (44%). PUK is the most common corneal finding in patients with Wegener's granulomatosis (WG), polyarteritis nodosa, and a well recognized manifestation of relapsing polychondritis. On the other hand it is rare in patients with systemic lupus erythematosis.

There is probably a common underlying mechanism in all patients with PUK who have CVD. The current theory is that immune complexes are deposited in the limbal vessels, activating the classic pathway of the complement system, which results in chemotaxis of inflammatory cells to the peripheral cornea. These cells release proteases and collagenases which destroy the corneal stroma. Inflammatory cells of the adjacent conjunctiva have been shown to release cytokines, which stimulate keratocytes to produce enzymes that can also degrade the stroma. Although serum antibodies to corneal epithelial antigens has been demonstrated in these patients, its pathogenetic role is not yet clear.

The currently recommended treatment for PUK in CVD includes resection of the conjunctiva adjacent to the corneal ulcer (which is believed to remove a source of inflammatory cells and cytokines from the ulcerated cornea), ulcer debridement and tissue adhesives for pending or small (up to 2 mm) corneal perforation, followed by placement of a bandage contact lens. Penetrating or lamellar keratoplasty and corneoscleral grafts are indicated when the corneal defect is large or when the tissue adhesive treatment has failed. The above treatment options have only temporary results and recurrences are common unless the underlying inflammatory response is controlled.

It is important to note that topical corticosteroids are contraindicated in patients with PUK and CVD since they may increase the likelihood of corneal perforation by inhibiting the production of collagen. Medications that inhibit the topical production of collagenase or the production of collagenase synthetase, such as topical 1% medroxyprogesterone, 20% acetylcysteine, or oral tetracycline, may be of limited benefit.

Although systemic corticosteroids have been effective in the treatment of PUK, the response to systemic cytotoxic immunosuppressive agents appears to be better in terms of ulcer healing rates as well as in overall survival of these patients (especially the WG patients). Cyclophosphamide (2 mg/kg/day) is the first choice, especially for the WG patients, followed by azathioprine (2 mg/kg/day), methotrexate (7.5-15 mg/wk) and Cyclosporin A (5 mg/kg/day).[4]

NEWER AND EXPERIMENTAL TREATMENT OPTIONS IN AUTOIMMUNE UVEITIS PATIENTS

Most patients with sight-threatening posterior uveitis (inflammation involving the retina and choroid) eventually receive systemic medications to control their disease. These medications may offer a significant visual improvement, at least in the short term, but the associated side effects can be severe. Corticosteroids remain the mainstay of treatment and can be administered by mouth, intravenously, locally by periocular injection or topically.[6] Experimental studies in rabbits with induced uveitis suggest that slow-release devices containing dexamethasone are effective in the treatment of severe panuveitis. When the inflammation is re-induced the treated animals show less active inflammation. After antigen rechallenge with the device in situ, inflammation in experimental eyes is still less than in controls.[7]

When steroids are ineffective, or contraindicated, additional therapy is required. Methotrexate (MTX) can be given at low doses in a one dose weekly pulse (12.5 mg/wk or less). At this dose it is associated with fewer side effects and is more effective in treating patients with inflammatory pseudotumor, scleritis or orbital myositis. It is less effective in patients with intraocular inflammatory activity or systemic vasculitis such as that associated with WG, or polyarteritis nodosa. Reported side effects include mild elevation of transaminases, transient mucositis, partial alopecia and vomiting. Caution is required in patients with preexisting renal or hepatic disease.[6]

Cyclosporin A and FK506 are agents that suppress an early stage in T cell activation by blocking nuclear binding factors involved in lymphokine gene transcription.[7] FK506 has been used mainly in Japan to treat patients with Behcetís disease, Vogt-Koyanagi-Harada syndrome and refractory uveitis.[6] A new drug entering this group is rapamycin. The concern with any of these drugs is the systemic toxicity associated with therapeutic doses and the fact that they do not specifically target the pathogenic cells.[7]

The role of vitrectomy (removal of part or all of the vitreous gel surgically) in the management of chronic posterior uveitis remains controversial. The goal of vitrectomy is to reduce the need for long-term immunosuppressive therapy, induce disease remission and prevent the long-term complications of chronic intraocular inflammation such as macular edema.[6]

It has been proposed that in T cell-mediated diseases, the pathogenetic cell is of the Th-1 type. It has been suggested that enhancement of the Th-2 component of the response against the antigen involved in the triggering of the autoimmunity should control the inflammatory process. Recombinant IL-4 and IL-10 are currently being studied as Th-2 inducing agents to control the development of experimental autoimmune uveitis.[7]

Oral administration of antigen is recognized as an effective method to achieve antigen-specific suppression without compromising the effectiveness of the immune system to respond to other stimuli. The use of oral administration of antigen as treatment for uveitis was first studied by Nussenblatt and colleagues. It is now well known that oral tolerance can be induced towards ocular antigens and their peptides.[8] Nussenblatt and colleagues recently reported the results of a phase I/II randomized masked trial which is the first study aimed to evaluate the effect and safety of the oral administration of retinal antigens as a treatment of ocular inflammation. The study concluded that patients given S retinal antigen were more likely to be tapered off their chronically administered systemic immunosuppressive therapy compared with patients given placebo.[9]

Gene therapy may become a major therapeutic modality in the treatment of ocular inflammation. Taking into account that the ideal method for gene insertion has yet to be found, several options for treating patients with posterior uveitis can be considered. Immunosuppressive cytokine insertion is being explored and cytokines, such as transforming growth factor beta-1 and interleukin 10, have been used in ex-vivo experiments. Posterior inflammation can lead to apoptosis of photoreceptors. The insertion of factors to prevent the apoptotic process could be a helpful post-inflammatory treatment to preserve photoreceptors. It might also be possible to use genes in an effort to insert immunosuppressive agents into ocular cells, particularly those that do not require any post-transitional modification. Active interference with factors that participate in the activation or inhibition of expression is a major promising method to control intraocular inflammation. There is evidence that interferon-gamma can regulate genes of ocular tissues through specific transcription factors. Among the studied transcription factors is activator protein-1 (AP-1), which increases interleukin-2 expression. AP-1 can also block activated glucocorticoid receptors, thereby reducing the effect of steroids during an immune response.[8]

CONCLUSION

The mainstay of treatment for autoimmune diseases affecting ocular tissues has been the use of topical, local and systemic anti-inflammatory drugs. Steroids predominate as the treatment of choice, but nonsteroidal anti-inflammatory drugs and antimetabolites also play a significant role. Although effective to varying degrees, the generalized systemic side effects and lack of specificity of all these drugs for the disease process has limited their effectiveness.

The new challenge for a rational therapy in autoimmune-induced ocular inflammation is the clarification of the specific molecules causing disease. Once they are determined, treatment strategies aimed at the specific cellular process can be developed. The treatment strategy aimed at a specific cytokine or cell mediator will require us to go beyond conventional pharmaceuticals. Gene therapy, by necessity, will become a larger part of the therapy. Whether genetically engineered exogenous cells to produce a specific protein are introduced into the system, or whether the host cells are engineered to do the same, therapies will become more cellular and by definition more specific. Our future ability to treat these diseases will require an understanding of their molecular processes.

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New Perspectives in Glaucoma

S. Deborah Murphy, MD

For over a century, glaucoma has been considered to be an ocular disease characterized by elevated intraocular pressure (IOP) which led to damage of the optic nerve and thus to visual loss. Before the mid 1800s, acute glaucomas were treated by recommending patients control their emotions, avoid constipation, and avoid straining their eyes. Leeches were also considered useful. As unscientific as that sounds to physicians in the late 1900's, it is likely that in the next millenium our approach to glaucoma will differ just as significantly from our current approach.

In the English literature in 1835, MacKenzie documented his observations about glaucoma by saying that "the eyeball always in glaucoma feels firmer than natural." Tonometry, or the ability to measure eye pressure, became clinically possible and certainly added a measure of sophistication over palpating the globe through the eyelid to estimate the degree of firmness. The invention of the ophthalmoscope in 1850 allowed observations of cupping of the optic disc by clinicians. In 1857, vonGraefe's surgical iridectomy cured some cases of acute glaucoma (which we now call acute angle closure glaucoma), but was noted to be relatively ineffective in treating the more chronic symptom-free glaucomas (what we now refer to as open angle and secondary glaucomas). External filtration operations were later developed to treat chronic glaucomas. By the late 1800s, ophthalmologists firmly held that elevated IOP was the hallmark of glaucoma. Control of IOP in acute glaucoma by peripheral iridectomy and control of some cases of the more chronic glaucomas by filtration surgery and miotic drops solidly defined glaucoma as a disease of elevated IOP to practicing physicians in that era.

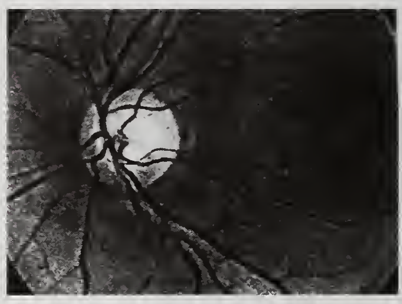
Our scientific understanding of the nature of glaucoma has expanded greatly due to the work of anatomists, basic scientists and clinicians aided by the tremendous technologic advances of the last several decades. The definition of glaucoma has evolved to the point that glaucoma is now considered to be an optic neuropathy characterized by loss of retinal ganglion cells (RGC) and their axons producing an excavated appearance of the optic nerve head and loss of the nerve fiber layer of the retina and resulting in characteristic visual field defects. Elevated IOP is perhaps the most important risk factor for the development of and progression of glaucoma, but it is not the disease itself. Other risk factors include a family history of glaucoma, advancing age, race (African-American), ocular trauma, and to a lesser extent myopia, hypertension, diabetes, steroid use and a history of shock or severe anemia.

Abbreviations Used:

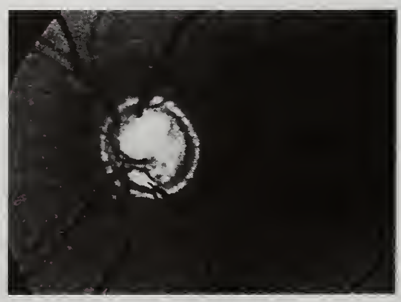
CNS	central nervous system
IOP	intraocular pressure
MFERG	multifocal electroretinography
RGC	retinal ganglion cell
SITA	Swedish interactive thresholding algorithm
SWAP	short wavelength automated perimetry

Clinically, there are many subtypes of glaucoma. Some are much better understood than others. For example, the mechanisms of ghost cell glaucoma, pigment dispersion glaucoma, pseudoexfoliative glaucoma, neovascular glaucoma, and angle closure glaucoma, to name a few, are far better defined than is primary open angle glaucoma. Primary open angle glaucoma is defined as a disease of elevated IOP in which characteristic optic nerve and visual field defects develop with no identifiable cause found by history, slitlamp examination, and gonioscopy. The term "primary" suggests we know the mechanism, but "idiopathic" open angle glaucoma would be more accurate.

There appears to be a relatively wide range in individual optic nerve susceptibility to IOP. For example, some patients can run IOPs consistently in the high 20's and never develop glaucomatous nerve damage. We call these patients ocular hypertensives or glaucoma suspects. Others develop characteristic damage with IOPs consistently in the statistically normal range of IOP (8 to 21 mmHg). These are the so-called normal tension or low tension glaucoma patients. Normal tension glaucoma is largely a diagnosis of exclusion. It is now felt that 20-30% of glaucoma may be normal tension glaucoma. It is not clear at this time whether the pathologic findings in eyes with normal tension glaucoma are the same as those in glaucoma associated with high IOP. Further, it is not clear that the treatment should be entirely the same. The Collaborative Normal Tension Glaucoma

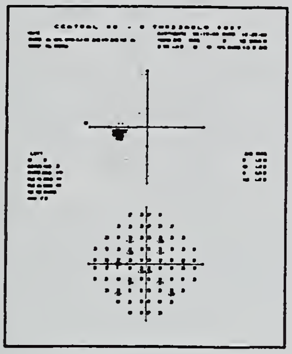


Normal optic nerve



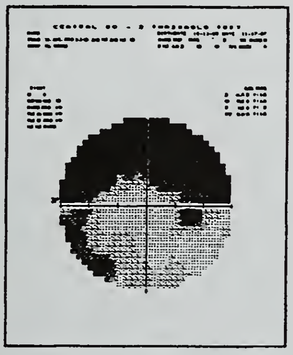
Glaucomatously cupped optic nerve

Figures 1a and 1b. Ophthalmoscopic view of normal optic nerve and nerve with advanced glaucomatous cupping.



Normal visual field

A printout of the visual field of a normal left eye. The dark area is the "blind spot" and occurs in all eyes.



Damaged visual field

The visual field of a damaged right eye. The dark area of visual loss was caused by glaucoma. The central area continues to have normal vision.

Figures 2a & 2b

Study Group just completed a study that established that IOP, even though it is not elevated, is an important part of normal tension glaucoma, and reducing pressure improves the outlook for these patients.

Before treatment of glaucoma may be undertaken, the diagnosis has to be made. We have come a long way from digital palpation of the eye. We can readily measure the IOP with a tonometer. We can observe the optic nerve and nerve fiber layer with the ophthalmoscope, but skilled judgment is required to detect early changes (Figure 1). High quality stereoscopic nerve head photography and nerve fiber layer photography are helpful in documenting a baseline to follow for change. More expensive and sophisticated means of documenting the nerve head exist such as automated stereo evaluators, digital optic nerve head analyzers, confocal scanning laser ophthalmoscopes and nerve fiber analyzers. These newer, expensive technologies are exciting, but in some cases, normative data and what constitutes a "significant" degree of change have yet to be adequately defined.

Visual field testing has also undergone several generations of change. Automated perimetry is routinely employed to help diagnose and follow glaucoma (Figure 2). Visual field testing is one of the ways we measure optic nerve function. Early visual field defects are generally not detected by patients and occur long before decreased visual acuity, as measured on the Snellen chart, develops. Yet 40-50% of optic nerve tissue can be lost before current visual field testing techniques can pick up defects. Automated visual fields test differential light sensitivity, and low patient reliability in taking this test can make useful interpretation impossible. Early in the disease process, damage to the RGC may go undetected because healthy neighboring cells with intact receptive fields may overlap the damaged cells' field. Strategies to minimize patient fatigue and to minimize receptive field overlap are being developed to help detect the glaucomatous disease process at an earlier stage. Frequency doubling technology promises to help in screening for glaucomatous field defects because it is relatively inexpensive, simple to use, and quick. Its sensitivity in following glaucomatous defects for progression has yet to be established. SWAP (short wavelength automated perimetry) may detect glaucomatous visual field defects 4-5 years earlier than current methods, but subjectively it is difficult for patients to perform, it is affected by the presence of cataracts, and normative data is not fully available. SITA (Swedish interactive thresholding algorithm) shortens testing time and, therefore, lessens patient fatigue, but visual field defects may not be as large or as dense as defects noted currently by threshold perimetry. MFERG (multifocal electroretinography) thus far is a research tool. It eliminates patient response, and in its current form takes about 8 minutes per eye and thus is relatively quick. It attempts to provide an objective, quantitative assessment of the visual field and looks promising.

It is possible that we may someday have the technology to count nerve fibers in specific areas of interest in the retina. Once normative data were known, the "count" could be followed and the diagnosis of glaucoma could be made early, certainly before visual field loss. This technology could also be used to monitor the effectiveness of treatment.

Perhaps the next millenium will also see genetically based diagnostic tests. Recently, at the annual meeting of the Human Genome Organization, it was reported that a defect in the TIGR gene accounted for about 25% of the open angle glaucomas. Although the exact percentage has been disputed by other scientists, there does appear to be some relationship between the TIGR gene and glaucoma, but scientists are still learning how the TIGR gene and its protein relate to the glaucomatous process and the health of the eye's drainage system. Some researchers feel TIGR protein may clog the trabecular meshwork (the drainage system of the eye) and cause increased IOP by that mechanism. They claim to have detected an over expression of the TIGR protein in the trabecular meshwork of some patients with normal tension glaucoma, some with pseudoexfoliative glaucoma, and up to 50% of the open-angle glaucoma cases they studied. In addition to exploring the development of a simple blood test to search for TIGR mutations, researchers hypothesize retinal gene therapy may also be possible in the future. This research, in its infancy, has exciting implications.

Ocular blood flow is considered by some investigators to have a relationship to glaucoma.[3] We are just beginning to develop useful tools to measure ocular blood flow. Color doppler imaging is used to study retrobulbar blood flow. (Figure 3). Scanning laser ophthalmoscopes can measure aspects of optic nerve and retinal blood flow. The

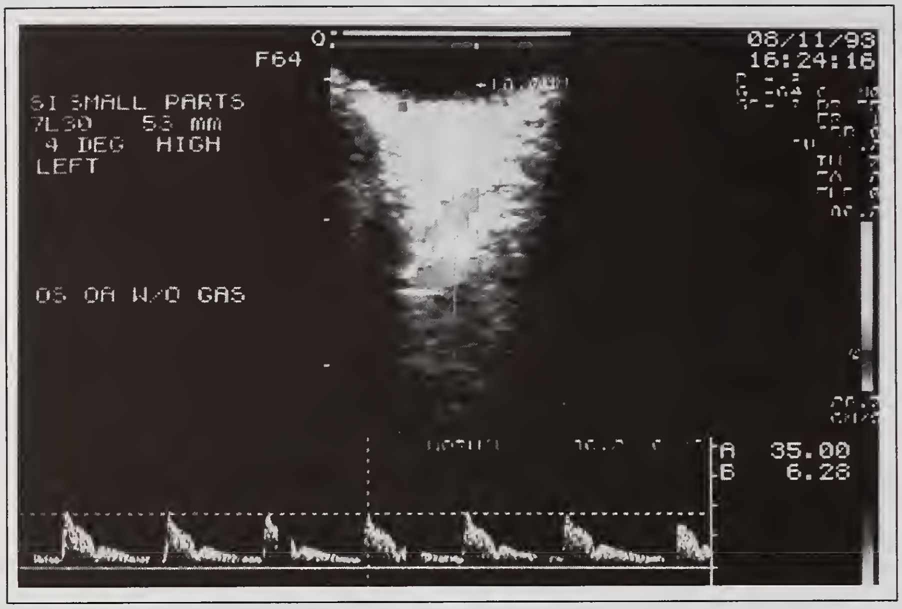


Figure 3. Example of an image from color Doppler imaging of retrobulbar vessels. The Doppler frequency spectra for the aphthalmic artery is shown as the waveform at the bottom of the figure. The corresponding peak systolic velocity and diastolic velocity and resistive index as determined from the waveform are shown on the bottom right.

Heidelberg retinal flowmeter takes tomographic scans through the optic nerve at various depths and constructs maps through the nerve to evaluate blood flow. Research in ocular blood flow is at an early stage, but studies thus far have suggested lower ophthalmic artery systolic velocities in glaucoma patients and decreased blood flow to the retina in glaucoma patients compared to age-matched normal patients. Whether these results will be substantiated remains to be seen, and the clinical significance has yet to be elucidated.

Mechanical factors seem to influence at least certain types of glaucoma.[3] Several types of acute glaucoma result in angle closure. Development of ultrasound biomicroscopy allows 50 micron resolution for cross-sectional imaging of the structures of the anterior segment of the globe. (Figure 4) This has proven useful in understanding and defining treatment strategies for certain glaucomas.

The ultimate goal in treating glaucoma is not to lower IOP, but to preserve vision and, thus, the quality of life. Medical, laser, and surgical means are all used.

Current medically oriented glaucoma treatment has expanded by many new classes of drugs but continues to address glaucoma primarily by lowering IOP. The traditional drug classes of cholinergic agents, sympathomimetics, systemic carbonic anhydrase inhibitors, adrenergic agonists, anticholinesterase agents and hyperosmotics have been added to by selective and non-selective betablockers, alpha agonists, topical carbonic anhydrase inhibitors and prostaglandins. As our medical armamentarium has expanded, so too has the scope of potential systemic side effects. Closer collaboration between ophthalmologists and primary care physicians has resulted.

Clinical trials are investigating new drugs, some of which are still aimed at reduction of IOP. For example, hypotensive lipids lower IOP and stimulate the contraction of certain PGF2 alpha-sensitive smooth muscle preparations as do the prostaglandins. However, they apparently do not stimulate or interact with previously described prostaglandin receptors.

Fortunately, as our scope of understanding the mechanisms of visual loss in glaucoma has expanded, so too drug research is looking beyond IOP reduction into neuroprotection and enhancing ocular blood flow. In the next millenium, it appears that the medical treatment of glaucoma will expand beyond lowering IOP into maintaining a healthy optic nerve and thus preserving vision from entirely new directions.

Better knowledge of the biochemical pathways involved in neuroprotection is needed and should soon be a key to the development of new drugs for the treatment of glaucoma. This area of research is so exciting because modifying IOP is currently the only medical treatment available to try to prevent and to arrest optic neuropathy and ganglion cell loss in glaucoma patients. This is a less than satisfactory approach because some patients never have demonstrable elevations in IOP and others continue to experience visual field loss long after normalization of their IOP. Researchers have felt that the continuing neuronal degeneration might, therefore, be the result of mediators produced by or associated with the degenerating neurons rather than from the primary risk factors that led to the degeneration in the first place. Basic science research in CNS suggests that "secondary degeneration" may be a related phenomenon to what goes on in glaucoma. CNS research has shown that regardless of some types of primary insult (e.g., ischemic, mechanical or degenerative disease) damage spreads beyond the directly injured neurons to adjacent neurons. The primary damage leads to changes in the extracellular environment, including changes in ion concentration, elevated levels of free radicals, release of neurotransmitters,

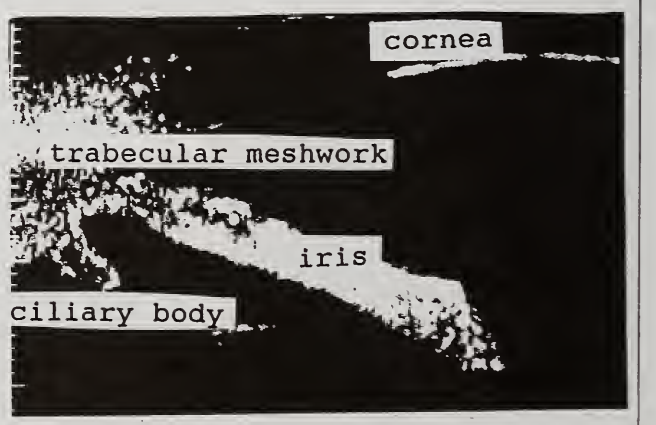


Figure 4. Ultrasound biomicroscopic image of a normal eye.

depletion of growth factors, and immune system changes. It is theorized that some or all of these changes trigger secondary degeneration.[4]

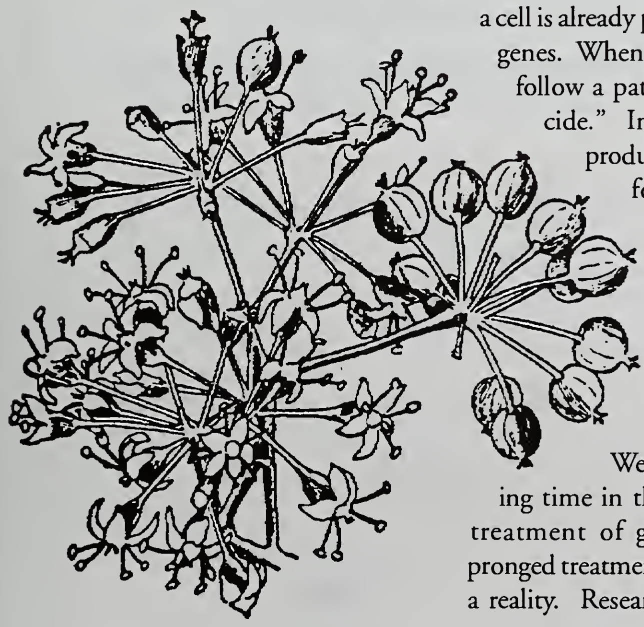
These findings are the basis of the concept of neuroprotection. The objective of neuroprotection is to employ pharmacologic or other means to regulate the hostile environment created by the primary insult and/or to supply the adjacent cells with the tools to deal with these changes. It is known that neurons from various CNS regions share many common properties; therefore, it is theorized that mediators involved in the propagation of secondary damage in the optic nerve in glaucoma may be similar to those that cause secondary degeneration in any part of the CNS. Researchers cite as support the fact that they have observed the presence of the excitatory amino acid glutamate in the vitreous body of glaucomatous eyes at concentrations that are potentially toxic to retinal ganglion cells. Glutamate is already considered a hypothetical cause of cytotoxicity in brain injury and brain disorders leading to secondary degeneration. Researchers are investigating several ways to modify the cellular response to glutamate.

Also under the umbrella of the neuroprotective drugs is a strategy to supply neurotrophins to the ganglion cells of the retina.[5] Neurotrophins are substances needed by neurons to survive. The lateral geniculate body or the superior colliculus in the brain produces the neurotrophin supply to the ganglion cells. These compounds are then transported along the axons to the cell body in the retina via axoplasmic flow. Researchers think that in glaucoma, the flow of neurotrophins may be impaired at the level of the lamina cribrosa (where the optic nerve enters the eye).[6] This neurotrophin deprivation may thus activate ganglion cell death. One area under study is to provide a different source or neurotrophins for the ganglion cells.

> *... glaucoma is now considered to be an optic neuropathy characterized by loss of retinal ganglion cells (RGC) and their axons producing an excavated appearance of the optic nerve head and loss of the nerve fiber layer of the retina resulting in characteristic visual field defects.*

The role of apoptosis in glaucoma is also of major interest.[7] Cells use apoptosis to maintain tissue homeostasis. Apoptosis means that the death of a cell is already programmed in its own genes. When signaled to do so, cells follow a pathway to commit "suicide." In apoptosis, cells must produce appropriate proteins for "suicide." Research is now directed at blocking apoptosis. Some of the compounds being explored inlcude lazaroids and protease inhibitors.

We are entering an exciting time in the early detection and treatment of glaucoma. A multipronged treatment strategy may soon be a reality. Research into disorders like stroke, Alzheimer's, amyotrophic lateral sclerosis and traumatic nerve injury may soon provide some answers to long-standing questions clinicians have had about glaucoma.



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The Foundation for Glaucoma Research allowed use of Figure 2.

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Rhode Island
Quality Partners, Inc.

Northeast Consortium Forms to Improve Prevention & Treatment of Pressure Ulcers in Long-Term Care Facilities

Prevention and Treatment of Pressure Ulcers in Long-Term Care Facilities

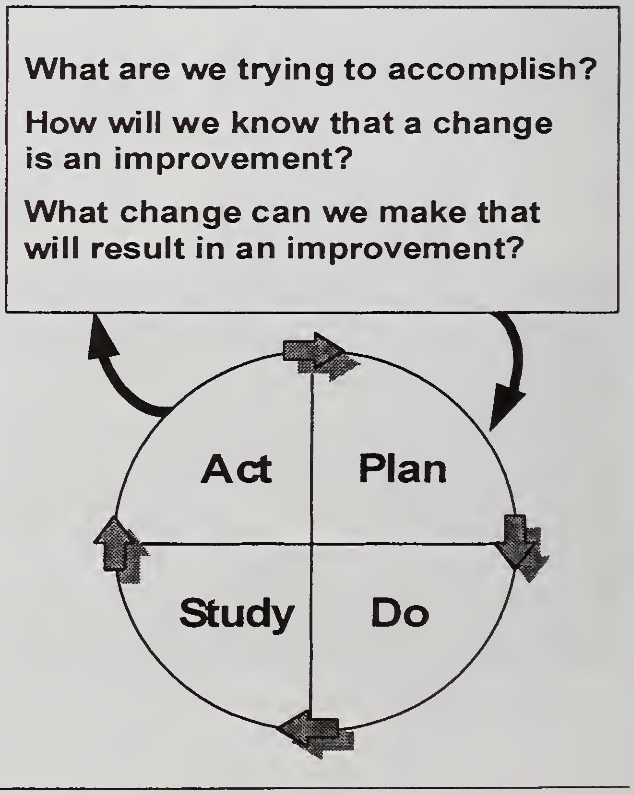
What do Rhode Island, New Jersey and Pennsylvania have in common? All three states have joined together and formed the Northeast Quality Improvement Consortium to improve the prevention and treatment of pressure ulcers in long-term care facilities.

This multi-state quality improvement initiative is truly unique, from the quality improvement methodology to the collaborators involved in the project. For the first time, the Boston, New York, and Philadelphia regional offices of the Health Care Financing Administration (HCFA), the peer review organizations (PROs) and state survey agencies in Rhode Island, New Jersey, and Pennsylvania are combining resources and expertise to improve care through implementation of continuous quality improvement (CQI) programs in long-term care facilities.

Since 1993 when HCFA established the Health Care Quality Improvement Program to better serve Medicare beneficiaries, peer review organizations throughout the country have worked on national and state-specific quality improvement initiatives, primarily in the acute care hospital setting. Today, HCFA has expanded the scope of this quality improvement initiative to other healthcare settings.

In Rhode Island, nine long-term care facilities have volunteered to be part of the Consortium and to work with HCFA (Boston Regional Office), Rhode Island Quality Partners (PRO), and the Rhode Island Division of Facilities Regulation. The participating facilities will implement CQI programs to improve the prevention and treatment of pressure ulcers among their residents. These efforts are based upon the clinical guidelines for pressure ulcer prevention and treatment developed by the Agency for Health Care Policy and Research and the American Medical Directors Association.

The Plan-Do-Study-Act improvement model (PDSA - See Figure 1) that will be used throughout this project will help facilities set aims (Plan), form teams to address specific aims (Do), establish quality improvement measures (Study), and identify opportunities for improvement (Act). Once facilities have determined which processes could be improved, the Plan-Do-Study-Act improvement model will enable the facilities to test the change on a small scale and to modify the process if necessary, before the change is adopted by the entire facility.


What are we trying to accomplish?
How will we know that a change is an improvement?
What change can we make that will result in an improvement?
Act
Plan
Study
Do

To assist the facilities participating in this improvement project, two experienced nurses in long-term care and quality improvement will serve as "facilitators" and meet regularly with the project teams. Throughout the project, team members will also be able to consult with medical experts in the field of pressure ulcer prevention and treatment.

This pilot project will give facilities experience in continuous quality improvement that, hopefully, can be applied to other health conditions and, ultimately, improve the health status of all long-term care residents.

Thus far, representatives from Rhode Island's collaborating facilities attended two workshops on continuous quality improvement and the care and treatment of pressure ulcers. Leaders in quality improvement including David Gifford, MD, MPH, clinical coordinator for Rhode Island Quality Partners (RIQP); and Joseph Lapenta, performance improvement administrator for Eleanor Slater Hospital, presented at each workshop. Additionally, participants worked in groups on a variety of case vignettes to practice team building and CQI skills.

To determine how the facilities currently perform on the prevention and treatment of pressure ulcers, each project team used a standard set of data collection tools that focused on risk assessment for pressure ulcer development, care plans, and implementation of care plans for residents at-risk for pressure ulcers. Throughout the data collection process, PRO facilitators Diane D'Ambra, MS, RN, and Pamela McCue, RN, MSN, provided ongoing training and support.

Following an analysis of baseline data, RIQP will hold a workshop in early 1999 for CQI team members from each facility to share experiences and discuss possible changes in their facility to improve pressure ulcer prevention and treatment.

* * *

This article was reprinted from the December 1998, Rhode Island edition of *Pressure Points*, newsletter of the Northeast Quality Improvement Consortium. For more information on this quality improvement initiative, please contact Project Coordinator Jennifer M. Lewis, RN, by phone: (860) 632-6343, fax: (860) 632-5865, or email: ctpro.jlewis@sdps.org.

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Health by Numbers

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Edited by Jay S. Buechner, PhD, and William J. Waters, Jr., PhD

Visual Impairment Among Rhode Island Adults

Jay S. Buechner, PhD

Partial or total impairment of vision is a disabling condition that can occur as a result of congenital defects, acute or chronic diseases, injuries, or aging. Those who experience loss of vision almost always suffer some level of functional limitation in such areas as their mobility, their employment, and their ability to care for themselves and live independently. Survey data for Rhode Island from 1996 have been analyzed to establish the prevalence of visual impairment in the state's adult population and the impact of this condition on employment status.

Methods

In 1996, the Rhode Island Health Interview Survey was performed by telephone and obtained information on all members of participating households, including demographic, social and economic characteristics, general and specific measures of health status and functional limitation, measures of utilization of health services, measures of access to health care, and other items. The survey covered 6,583 persons in 2,580 households. All persons ages 18 and older were asked whether they had any difficulty seeing the letters in ordinary newspaper print, even when using their glasses or contact lenses, and, if so, whether they could see such print at all. From these questions, a three-level scale of visual impairment was constructed: unable to see newsprint, difficulty seeing newsprint, and no difficulty seeing newsprint. Persons in the first two categories were considered to have a visual impairment at some level.

Results

Among adult respondents, approximately 4.0 % had difficulty reading newsprint and another 0.7% could not read newsprint at all, so that slightly under one in twenty (4.7%) Rhode Island adults experience some visual impairment. Applied to the estimated population of Rhode Island in 1997, this percentage translates into approximately 35,400 visually impaired adults in the state. Comparable survey data for the United States for 1993 provide a prevalence estimate among adults for this impairment of 4.7%, identical to the 1996 estimate for Rhode Island.[1]

The prevalence of vision impairments, both partial and total, rose sharply with age. (Figure 1) Persons ages 65 and older were most likely to have such impairment, with nearly one in ten (9.4%) being affected. The effects of age were seen more strongly among those with total impairment, where the prevalence among persons ages 65 and older was ten times that for persons ages 18 to 44 years. For partial impairment, the prevalence among the elderly was less than four times the prevalence among young adults.

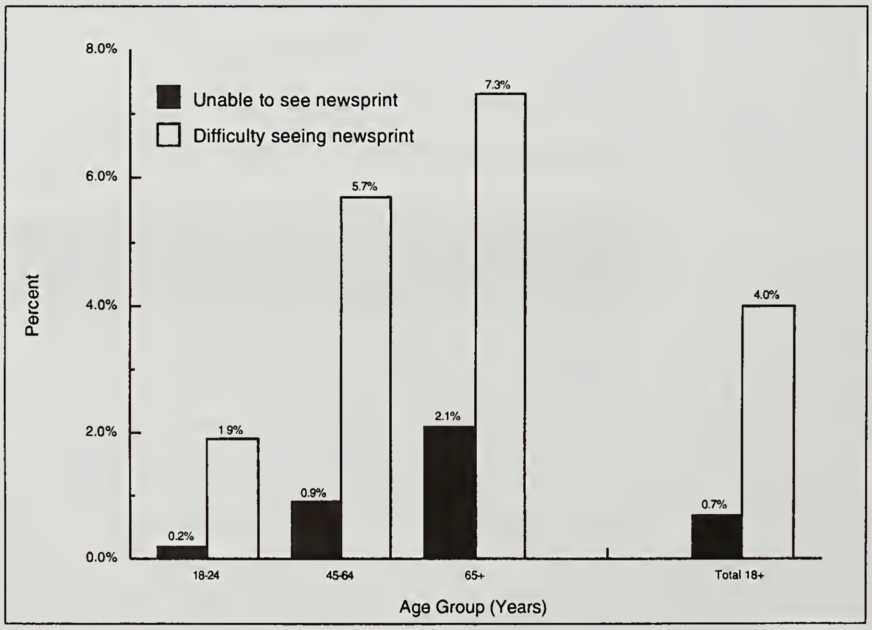

Figure 1. Prevalence of Vision Impairment, by Age Group and Level of Impairment, Ages 18 and Older, Rhode Island, 1996.

Women were somewhat more likely to be reported as having impaired vision (5.3%) than men (4.0%). (Figure 2) This gender differential persisted through all adult age groups, being greatest among persons ages 65 and older (46% higher prevalence), but also substantial among those ages 18 to 44 (27% higher) and those ages 45 to 64 (15% higher).

Having a visual impairment was a strong determinant of employment status for the surveyed population ages 18 to 64 years. Among persons with an im-

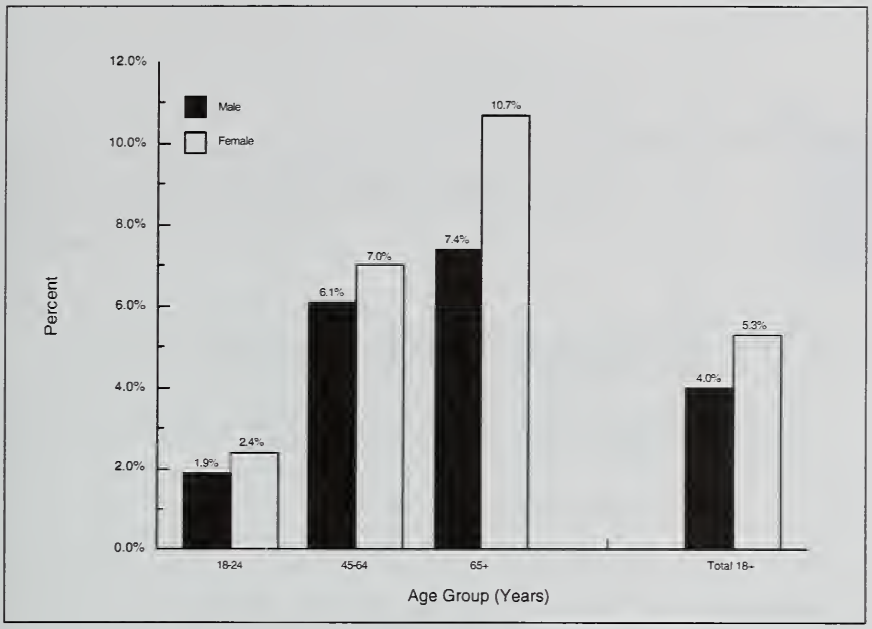

Figure 2. Prevalence of Vision Impairment, by Age Group and Sex, Ages 18 and Older, Rhode Island, 1996.

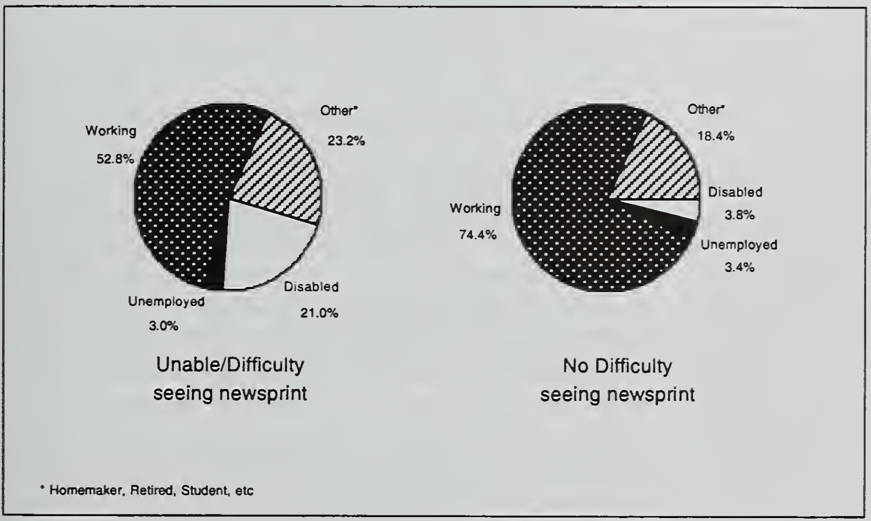

Figure 3. Employment Status by Presence/Absence of Vision Impairment, Ages 18 through 64, Rhode Island, 1996.

pairment, approximately 53% were employed and 3% unemployed, with the remaining 44% not in the labor force. Among those without an impairment, only 22% were not in the labor force. (Figure 3) The difference between the two groups was primarily in the proportions who were permanently or temporarily disabled - 21% of those with a visual impairment versus 4% of those with no impairment.

Discussion

Many Rhode Island adults suffer from visual impairment at a level that affects their ability to read ordinary newspaper print. Although the prevalence of this impairment is highest among those ages 65 and older, there are a significant number of working-age persons with vision problems. Despite their vision problems, a majority of these younger persons are working, and many others are going to school, keeping house, or otherwise active and functioning. Nevertheless, having a visual impairment often leads to being unable to work due to the resulting disability. The data presented here do not take into account the many other potential limitations of activity for those whose vision is impaired.

Jay S. Buechner, PhD., is Chief, Office of Health Statistics, and Clinical Assistant Professor of Community Health, Brown University School of Medicine.

References

1. Benson V and Marano MA. *Current estimates from the National Health interview Survey, 1993.* National Center for Health Statistics. Vital and Health Statistics 10(190). 1994.

NORMAN M. GORDON, M.D.
450 VETERANS MEMORIAL PKWY, EAST PROVIDENCE R.I. 02914
401-431-1860

AND

VLADISLAV ZAYAS, M.D.
ONE RANDALL SQUARE, SUITE 410, PROVIDENCE, R.I. 02904
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PUBLIC HEALTH BRIEFING

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

The Tobacco Settlement: Briefing for Physicians

Patricia A. Nolan, MD, MPH

BACKGROUND

The recent tobacco litigation by the States' Attorneys General began in May, 1994, with a lawsuit brought by the State of Mississippi against the tobacco industry. Mississippi sought to recover medical and ancillary costs incurred by the State for persons with diseases related to the use of tobacco, and asked for an injunction against marketing tobacco products to children. In a little over two years, 14 states followed Mississippi; another 25 followed shortly thereafter. Then Puerto Rico and a number of municipalities also sued. Although charges brought against the tobacco industry varied from place to place, a major goal of every suit was recovery of tobacco-related medical costs incurred by the government.

Faced with the specter of almost 50 simultaneous lawsuits, the tobacco industry opened negotiations with the states' Attorneys General in April, 1997, to seek a nation-wide settlement with all litigants. By June, 1997, the parties had roughed out a deal requiring the passage of special Congressional legislation. A month later, after careful scrutiny by a special task force, President Clinton publicly rejected one part of the agreement which limited the FDA's ability to regulate nicotine. Later, the White House demanded three significant changes in the proposed settlement. Former FDA Commissioner Kessler and former Surgeon General Koop weighed in against the proposed settlement as too friendly to the tobacco industry. Ultimately, the settlement fell apart in the spring of 1998 during negotiations in the Senate, when the industry walked out.

Negotiations between the states' Attorneys General and the industry resumed this spring, using a formula that avoided Congressional approval for adoption. A revised settlement was proposed in November, 1998, and was ultimately adopted by the industry, 46 states, the District of Columbia, and five territories. (Florida, Minnesota, Mississippi, and Texas had settled their lawsuits separately.)

SYNOPSIS OF THE SETTLEMENT

The settlement provides $206 billion to the states over 26 years, then continues providing payments in perpetuity. Rhode Island will receive about $1.4 billion in 26 years, then about $50 million per year thereafter. About half may be claimed by the federal government under a law which requires states to refund the federal share of funds recovered for Medicaid, although efforts are underway at the federal level to avoid this.

Abbreviations Used:

CDC	Centers for Disease Control and Prevention
FNA	fine needle aspiration
NBCCEDP	National Breast and Cervical Cancer Early Detection Program
WCSP	Women's Cancer Screening Program

The settlement bans promotion of tobacco products that directly or indirectly target youth. It bans the use of cartoon characters in ads, and it bans all billboard and transit advertising. Still allowed is point of purchase advertising, including outdoor advertising of modest dimensions. The settlement also bans sponsorship of most sports and cultural events. It allows each tobacco company one brand name sponsorship of a major event each year, such as the Winston Cup.

Free tobacco samples, merchandise with brand name logos, and distribution of unpackaged cigarettes are banned. (Free samples may be distributed in settings which exclude persons under age 18.) The minimum pack size for cigarettes is 20 until the year 2001. In Rhode Island, it is already illegal to sell cigarettes in less than 20-unit packs.

The settlement restricts the tobacco industry's lobbying, prohibiting tobacco industry opposition to laws which limit youth access to tobacco, and grants public access to tobacco industry documents on the internet.

The settlement includes several other resources in addition to the $206 billion awarded directly to the states. It sets aside $50 million nationally to fund enforcement of the settlement by the states' Attorneys General. It gives $25 million for 9 years to create a National Foundation to study programs to reduce teen smoking and substance abuse. It provides $250-300 million per year for five years to create a national tobacco education fund, with the likelihood that this fund will continue in perpetuity as well.

The awarding of funds is contingent upon final approval of the settlement by at least 80% of the 46 states involved in the settlement, but no later than July, 2000. The deal expires on December 31, 2001, for any state unable to win a consent decree from the court.

Role of the General Assembly

Funds awarded to the State of Rhode Island from the tobacco settlement go to the General Fund for appropriation by the General Assembly. The Assembly is free to allocate the money in any way it sees fit, although the Rhode Island consent decree entered on 17 December 1998 provides that the money is intended to be held in trust, for use on health-related issues.

20% for Tobacco Control!

The settlement gives us a unique opportunity to prevent tobacco use and to help smokers quit. We can use these resources to reverse the upward trend in teen smoking recently experienced in Rhode Island. Proven approaches are available to do both, but need substantial support to achieve desirable results statewide. The New England Division of the American Heart Association, in collaboration with the New England Division of the American Cancer Society and the American Lung Association of Rhode Island, recommend that 20% of the settlement funds in Rhode Island go to tobacco use prevention and cessation programs. These include large-scale counter-advertising campaigns, tighter law enforcement, population-based approaches to smoking cessation, and scientific evaluation of the effects of prevention and cessation efforts:

- Large-scale counter-advertising combats the tobacco industry's relentless pursuit of our children through ubiquitous community advertising. The California and Massachusetts campaigns caused major reductions in tobacco consumption.
- Tighter law enforcement prevents the illegal sale of tobacco products to minors. Keeping llegally obtained tobacco out of the hands of children is an effective way of preventing addiction.
- State-of-the-art approaches to smoking cessation really help smokers to quit, and to avoid smoking again. State-of-the-art approaches run the gamut from brief clinical interventions designed for the physician's office to population-based interventions designed for widespread application. Researchers at the Brown University School of Medicine and the University of Rhode Island have pioneered cessation approaches used all over the world. With sufficient funding, these experts could organize a statewide cessation program that really worked to lower smoking rates in Rhode Island.
- Scientific evaluation of tobacco control interventions will assure that they achieve their intended impact.

Useful websites on the tobacco settlement:
http://headlines.yahoo.com/Full_Coverage/US/Tobacco_Settlement/Related_Web_Sites.html
http://www.tobaccosettlement.org/
http://www.cancer.org

Patricia A. Nolan, MD, MPH, is Director of Health.

Brown University AIDS Program

PRIMARY PROVIDER AIDS EDUCATION PROGRAM

March 10 & 17, 5:00 - 9:00 pm

The Brown University AIDS Program, with the assistance of the New England AIDS Education and Training Center, is sponsoring a two-part program to train primary care providers in issues surrounding AIDS and Human Immunodeficiency Virus (HIV) infection. Our goal is to facilitate comfort in the evaluation and management of individuals with HIV infection, and to increase knowledge of resources available for appropriate triage.

Topics to be covered on March 10 include:

- Recognizing and Monitoring HIV Infection
- Infection Control Practices
  (One hour blood borne pathogen CME offered)
- Strategies for HIV Prevention

Topics to be covered on March 17 include:

- Antiretroviral Therapy Update
- Opportunistic Infection Management
- Psychosocial Impact of HIV Infection

Lectures on March 10 will be followed by clinical case discussions.

* * *

This schedule is subject to change.
Seating is limited & registration is mandatory.
To register and obtain a final program
call: 401 863-1725 or check our website at
www.brown.edu/Departments/BRUNAP/

* * *

The Brown University School of Medicine is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians.

The Brown University School of Medicine designates this activity for a maximum of 6.0 hours in Category 1 of the Physician's Recognition Award of the American Medical Association. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health
Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report
Provisional Occurrence Data from the Division of Vital Records

Vital Events

	Reporting Period		
	August 1998	**12 Months Ending with August 1998**	
	Number	**Number**	**Rates**
Live Births	1,119	13,378	13.5*
Deaths	753	9,851	9.9*
Infant Deaths	(6)	(102)	7.6#
Neonatal deaths	(5)	(79)	5.9#
Marriages	909	7,481	7.6*
Divorces	217	3,174	3.2*
Induced Terminations	376	4,801	358.9#
Spontaneous Fetal Deaths	58	955	71.4#
Under 20 weeks gestation	(51)	(876)	65.5#
20+ weeks gestation	(7)	(79)	5.9#

* Rates per 1,000 estimated population
# Rates per 1,000 live births

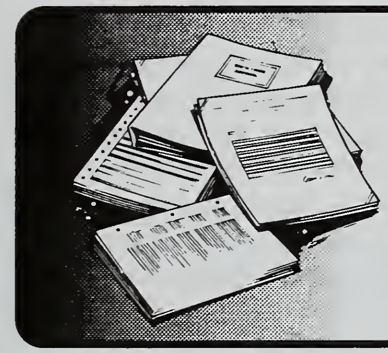
Underlying Cause of Death

	Reporting Period			
	February 1998	**12 Months Ending with February 1998**		
	Number (a)	**Number (a)**	**Rates (b)**	**YPLL (c)**
Diseases of the Heart	339	3,273	330.5	3,789.0
Malignant Neoplasms	166	2,464	248.8	7,089.5
Cerebrovascular Diseases	58	678	68.5	734.5
Injuries (Accident/Suicide/Homicide)	31	327	33.0	6,341.5**
COPD	62	463	46.8	362.5***

**Excludes two deaths of unknown age ***Excludes one death of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.
(b) Rates per 100,000 estimated population of 990,225
(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.



CLINICAL TRIALS DIRECTORY

Medicine & Health/ Rhode Island is pleased to launch this Directory. In Rhode Island, many researchers—hospital-based and community-based—are conducting clinical trials; but the channels of communication are not optimal. We intend this Directory of Clinical Trials to serve as an information clearinghouse for ongoing trials in the state. If you would like to list a trial, please contact: Joan Retsinas, Managing Editor
phone: (401) 272-0422
fax: (401) 272-4946
e-mail: JRetsinas@aol.com.

A Randomized, Double-Blind Study of MKC-442 Combined With Viracept® in Patients Who Are Epivir® + Retrovir®, Experienced and are Protease-Inhibitor and Non-Nucleoside Reverse Transcriptase-Inhibitor Naïve (MKC 303)

Sponsor: Triangle Pharmaceuticals

Purpose: The purpose of this study is to assess the usefulness of a new experimental non-nucleoside reverse transcriptase inhibitor (MKC-442) in combination with the protease inhibitor Viracept® (nelfinavir) and two nucleoside analogues (NRTIs), at least one of which you have never received before.

Principal Investigator: Karen Tashima, MD

Patients recruited: Patients must be at least 18 years of age. Patients must have taken 2 NRTIs for at least 16 weeks and have an HIV-1 viral load of at least 10,000 copies/mL; *or*, patients must have never taken HIV medications, and have a plasma HIV-1 RNA >50,000 copies/mL and CD4+ count <300 cells/mm3.

Intervention: Upon being accepted to participate in this study, nucleoside experienced patients will be asked to switch to Zerit® (d4T, stavudine) + Epivir® (3TC, lamivudine) or Zerit® (d4T, stavudine) + Videx® (ddI, dideoxyinosine). Patients who have not taken HIV medications will be asked to choose between two different dual NRTI regimens, 3TC + d4T or d4T + ddI. Viracept® (nelfinavir) at two different doses, has been chosen as the protease inhibitor to be prescribed in combination with active or placebo MKC-442 and the two chosen nucleosides.

Duration of Study: This is a 48 week clinical trial.

Phase: Phase II

Site(s): The Miriam Hospital Immunology Center

Remuneration for patients: $25.00 per scheduled visit.

Contact person: Joan Gormley, RN

Phone: 401-793-4396

Fax: 401-273-0285

e-mail: jgormley@lifespan.org

A Randomized, Double-Blind Study Of Mkc-442 Combined With Stavudine, Didanosine, And Hydroxyurea In HIV-Infected Patients Who Are Protease Inhibitor Experienced And Non-Nucleoside Reverse Transcriptase Inhibitor Naïve (Mkc-305)

Sponsor: Triangle Pharmaceuticals

Purpose: The purpose of this study is to offer another option for people who have failed protease inhibitor containing regimens.

Principal Investigator: Karen Tashima, MD

Patients recruited: Patients must be at least 18 years of age. Patients must be NNRTI naïve, and have a plasma viral load of more than 5,000 but less than 50,000 copies mL.

Intervention: The study drugs assigned will be based partly on the results of an HIV RNA test, and partly on the results of a random selection. Patients will receive the drugs stavudine (d4T), didanosine (ddI) and hydroxyurea (Hydrea). The experimental NNRTI, MKC-442 or its placebo (inactive pill) will be added to these drugs.

Duration of Study: This is a 48 week clinical trial.

Phase: Phase II

Site(s): The Miriam Hospital Immunology Center

Remuneration for patients: $25.00 per scheduled visit.

Contact person: Helen Sousa, LPN

Phone: 401-793-4771

Fax: 401-273-0285

e-mail: jgormley@lifespan.org

*** Zyban Collaborative Study**

Sponsor: National Institutes of Health; The Miriam Hospital, a Lifespan partner; and Butler Hospital, a Care New England partner

Purpose: The Zyban Collaborative Study is investigating the effects of the combination of Zyban, an FDA-approved medication for smoking cessation, and group counseling to assist with quitting smoking. It is anticipated that this combination of treatments will be particularly effective in the increasing number of "hard core" smokers.

Principal Investigator: Raymond Niaura, PhD, and Richard Brown, PhD

Patients recruited: This free research study is open to cigarette smokers who are at least 18 years old, in good health, smoke 10 or more cigarettes a day, and want to quit smoking.

Intervention: Participants have a 50/50 chance of being assigned the active medication Zyban versus a placebo; they will receive a free medical screening and 12 group counseling sessions.

Duration of study: Participation in the study will last up to one year and will include 12 group visits during a 3-month treatment period, and 3 follow-up visits at 2, 6, and 12 months after the start of treatment.

Phase: Subject recruitment will continue for approximately two-and-a-half years. Participant screenings are scheduled on a continuous basis; new groups begin every seven weeks. Evening groups are available.

Site(s): The Miriam Hospital and Butler Hospital

Compensation: Participants will receive up to $140 for completing all of the questionnaires and follow-up assessments after treatment ends. Abstinence from smoking is not required in order to be reimbursed.

Contact: The Lifespan Health Connection for further information and screening for participation in the study. phone (401) 444-4800. Dr. Elizabeth Lloyd with specific questions. phone (401) 793-3714,

e-mail: ELloyd@Lifespan.org

* **Congenital Estotropia Observational Study**

Sponsor: National Eye Institute of NIH

Purpose: Despite the common occurrence of congenital esotropia, prospective data on its early course are limited. Such data are needed to determine the earliest age at which surgery can be safely performed without concern that the esotropia is likely to resolve spontaneously. To provide these data, an observational study of congenital esotropia is being conducted the Pediatric Eye Disease Investigator Group, a group of about 150 pediatric ophthalmologists located through North America, formed to conduct clini-

Information for Contributors

Medicine & Health/Rhode Island welcomes submissions from members of the Rhode Island health care community. Submissions can fall into one of three categories:

Contributions

Contributions should report on an issue of interest to clinicians in the state: new research, treatment options, collaborative interventions, review of controversies. The maximum length of submissions is 2500 words; the maximum number of footnotes is 15. (The Journal is not the venue for an exhaustive literature review). Tables, charts, and figures should be camera-ready. Photographs should be black and white. (Slides are not accepted).

Creative Clinician

Clinicians are encouraged to submit brief (no more than 1200 words) descriptions of cases that defy texbook analysis. Photographs, charts and figures may accompany the case; footnotes should not exceed 6.

Point of View

The column gives readers an opportunity to share their perspective on any issue facing clinicians. The topic is broad: it could, for example, include ethics, health care policy, and/or relationships with patients. Maximum length is 1200 words.

The format of submissions is as follows:

The title page should include name, affiliation, address, phone, fax, and e-mail. A brief abstract should appear on a separate page. References should be numbered sequentially in the text, and listed separately at the end of the document (not the end of each page).

For Contributions and Point of View, please submit 4 hard copies of the document, with a disk (Microsoft Word or Text), to the managing editor: Joan Retsinas, PhD, 344 Taber Avenue, Providence, RI 02906.

For Creative Clinician columns, please submit 3 hard copies of the document, with a disk (Microsoft Word or Text), to Anthony Mega, MD, Miriam Hospital, Providence, RI 02906.

For additional information, please contact Joan Retsinas (phone: (401) 272-0422; e-mail: JRetsinas@aol.com).

Book Reviews

Medicine & Health/Rhode Island will review books authored by Rhode Island physicians. Publishers should send a copy for review to the managing editor.

cal research in pediatric ophthalmology.

Patients Recruited: patients, between 9 and 17 weeks of age, with a gestational age >= 37 weeks and birth weight > 2000 grams (4 lb, 6 oz), and who are neurologically and developmentally normal.

Intervention: an examination (identical to the pediatric ophthalmologist's usual routine, with no additonal procedures performed specifically for the study), and two follow-up visits, one 2-4 weeks after the first examination, the other when the child is between 28 and 32 weeks of age.

Site: office of David Robbins Tien, MD, 110 Lockwood Street, Suite 440, Providence, RI 02903

Contact: David Robbins Tien, MD, phone: (401) 444-7008; fax: (401) 444-4862. For more information about other participating pediatric ophthalmologists, contact the PEDIG Data Coordinating Center, (888) -79PEDIG

*A Multi-Center, Double-Blind, Placebo-Controlled, Parallel-Group, Dose-Ranging, Study for the Safety, Tolerability and Efficacy of S1B-1508Y in Parkinson Disease Patients who are Requiring but Not Receiving Dopaminergic Therapy

Sponsor: S1B1A Neurosciences and the Parkinson Study Group

Purpose: Joseph Friedman, MD, is conducting this trial to treat patients with early Parkinson's Disease. Patients will be treated with varying doses of a nicotinic agonist looking at both symptomatic motor effect and memory enhancement.

Patients Recruited: People with Parkinson's Disease not receiving Dopaminergic drugs. MMSE must be >24. May be on Eldepryl. No agonists, amantadine, antidepressants, or neuroleptics.

Intervention: varying doses of S1B-1508Y vs. placebo

Duration of study: 5 weeks

Phase: IIa

Site: Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

*A Multi-Center, Placebo-Controlled Trial of Melatonin for Sleep Distrubance in Alzheimer's Disease

Sponsor: Alzheimer's Disease Cooperative Study

Purpose: Brian Ott, MD, is conducting this trial to treat Alzheimer's Disease patients who experience sleep disturbances associated with Alzheimer's Disease. Two doses of melatonin will be used to treat sleep disturbances in order to lessen the burden on caregivers and family members.

Patients Recruited: Anyone 55 years or older with a diagnosis of probable Alzheimer's Disease experiencing sleep disturbances.

Intervention: Two doses of Melatonin vs. placebo

Duration of study: 12 weeks

Phase: III

Site: ALzheimer's Disease & Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860

Contact: Michael Pimental, MA, phone (401) 729-3752

*Earlier vs. Later Levo-dopa in Parkinson's Disease (ELLDOPA)

Sponsor: NIH and the Parkinson Study Group

Purpose: Joseph Friedman, MD, is conducting this trial to compare the effect of early or late treatment of Parkinson's Disease with L-Dopa to answer the question of whether L-Dopa slows or hastens the progression of Parkinson's Diease.

Patients Recruited: Patients must be 30 years or older, must be diagnosed with Parkinson's Disease within the last 2 years. No eldepryl, L-DOPA, amantadine, anticholinergics, antihistamines, antidepressants, or benzodiazepines for previous 30 days.

Intervention: varying doses of Carbidopa/Levodopa vs. placebo

Duration of study: 9 months

Site: Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860

Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

*A Phase III Multicenter, Double-Blind, Parallel-Group Placebo Controlled Study of the Effect of Riluzole 50 mg BID or 100 mg BID for Two Years on the Progression of Parkinson's Disease in 1050 Patients

Sponsor: Rhône-Paulena Rorer

Purpose: The purpose is to treat recently diagnosed patients with the drug Riluzole, in order to study the effect this drug has on delaying the progression of Parkinson's Disease.

Principal Investigator: Joseph H. Friedman, MD

Patients Recruited: Patients must have recently diagnosed Parkinson's Disease with symptoms present for no more than 3 years and must currently be receiving no medications to treat Parkinson's Disease.

Intervention: Two doses of Riluzole or Placebo

Duration of Study: 2 years

Phase: III

Site(s): Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Margaret C. Lannon, RN, MS; phone: (401) 729-3750

***Parkinson Disease Collaborative Study of Genetic Linkdate (PROGENI)**

Sponsor: NIH and the Parkinson Study Group

Purpose: The purpose is to explore genetic, environmental and other factors which may play a role in the development of Parkinson's Disease.

Principal Investigator: Joseph H. Friedman, MD

Patients Recruited: Looking for sibling pairs (brothers and/or sisters) who both have Parkinson's Disease or suspected Parkinson's Disease. Siblings need NOT live in Rhode Island.

Intervention: None

Duration of Study: One visit

Site(s): Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Margaret C. Lannon, RN, MS; phone: (401) 729-3750

***Treatment of Psychosis and Associated Behavioral Disturbances in Patients with Dementia**

Sponsor: a major pharmaceutical company

Purpose: The purpose is to determine whether treatment of persons with psychosis and behavioral disturbance related to Alzheimer's Disease or vascular dementia benefit from treatment with investigational antipsychotic drugs.

Patients Recruited: Patients must be greater than 40 years old and have hallucinations or delusions associated with dementia caused by Alzheimer's Disease or stroke.

Intervention: One of two investigational antipsychotic drugs, or placebo

Duration of Study: 28 weeks

Phase: III

Site(s): Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Michael Pimental, MA; phone: (401) 729-3752

***A Randomized, Double-Blind Placebo-Controlled Trial to Evaluate the Safety and Efficacy of Vitamin E and Donepezil HC1 (Aricept®) to Delay Clinical Progression to Alzheimer's Disease (AD) from Mild Cognitive Impairment (MCI)**

Sponsor: Alzheimer's Disease Cooperative Study and Pfizer/Elsai

Purpose: The purpose is to determine whether treatment of persons with mild forgetfulness can prevent the actual development of Alzheimer's Disease or slow the progression of Alzheimer's Disease

Principal Investigator: Brian R. Ott, MD

Patients Recruited: Patient must be 55 to 90 years of age and have memory difficulties that are not severe enough to interfere with carrying out one's usual daily activities (Mini-Mental=24-30). Must be willing to take only vitamin supplement provided.

Intervention: Multivitamin, Vitamin E, or Donepezil HC1

Duration of Study: 3 years

Phase: III

Site(s): Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Michael Pimental, MA; phone: (401) 729-3752

Letter to the Editor

[Re December 1998 issue]

The Rhode Island Blood Center (RIBC) appreciates the acknowledgement given to the contributions Ronald A. Yankee, MD, has made to the practice of transfusion medicine in Rhode Island and nationally in the December 1998 issue highlighting blood banking and transfusion medicine. His retirement after 20 years of dedicated service comes at a time when new challenges and opportunities face RIBC. Challenges of supplying the blood demands of our community may be met with the use of new technology and procedures such as apheresis, a method of blood collection with which Dr. Yankee was involved early in his career, which is also used for peripheral blood stem cell collections, an alternative to the traditional bone marrow transplant.

RIB is excited about the new opportunities...in this age of information. It is possible to link the informatics of various hospitals with the blood center to provide a more seamless continuity of care.

In addition, as Joseph D. Sweeney, MD, stated, the future of transfusion medicine in Rhode Island holds the routine removal of allogeneic leukocytes from all blood products as a goal to be implemented by the beginning of the next millenium. Continued development of tests on the blood supply lead to continued improvement on an already very safe blood supply. Also in the spring of 1999, RIBC will be implementing tests to detect nucleic acids of specific viruses (HIV, HCV)...

All of these advances wouldn't have been possible without the original vision, hard work, and dedication of Ronald A. Yankee, MD.

– Carolyn Te Young, MD

Medical Director, Rhode Island Blood Center

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Point of View

Why Didn't Our Doctor Tell Us About Hospice Sooner?

Edward W. Martin, MD

Referrals to hospice are often delayed until hours or days before the patient's death. Nationally, the median survival after admission to hospice was only 36 days in 1990.[1] Our experience locally is similar; in fact, length of stay in hospice has declined in the past few years.

The problems this creates for patients and families was detailed in the *Annals of Internal Medicine*.[2] In a letter to his wife's physician, a man asked why the physician had not told them about hospice sooner. The man detailed the stresses of caring for his dying wife alone without being able to understand what was happening. She was finally admitted to hospice on Friday and died on Sunday. This, unfortunately, is a fairly typical scenario; and families complain bitterly in follow-up interviews that referral came too late. Families report that they were left to manage the dying process alone and isolated.

It is unclear why this situation persists. Certainly the vast majority of physicians have worked with hospice programs in caring for their terminally ill patients and their families; yet physicians often don't discuss hospice with those patients and, many times, refer only when severe symptoms have developed and the patient has only days or hours to live.

One of the problems, I believe, is the misconception regarding the value of hospice or pre-death care to a terminally ill patient. We think it poor practice not to insure that a pregnant woman receives prenatal care. This care includes not only medical care, but also educational and emotional support. The physician who told the pregnant patient, "There is nothing more I can do for you" would be viewed as negligent. This is, unfortunately, often the message that terminally ill patients get. In the absence of uncontrolled symptoms, they are sent home to confront the dying process alone, with no formal support. The Rhode Island Health Department convened an expert panel on cancer treatment which identified a need for improved care of the dying in Rhode Island, and requested that an expert panel on palliative care be formed. One goal was that all terminally ill patients have access to hospice services. This will require several things of physicians:

1) Prognosis must be discussed openly and honestly with patients. While not taking away hope, patients have a right to expect information to assist them in planning how they will spend their final months of life.
2) The physician or designee must explain that hospice care is available should the patient and family wish to access it during the final months of life.

Physicians appropriately focus on the physical comfort of their patients. A patient with severe pain or uncontrolled nausea and vomitting cannot benefit from the other services they may receive from hospice. Such a patient will be hard pressed to benefit from time with the chaplain or social worker. Yet when families and patients talk about their experiences on hospice, it is generally not about their lack of pain or control of nausea. It is the support and care they receive from the members of the hospice team. The hospice nurse is often the first individual to sit with the patient and answer questions about the dying process. The chaplain may have offered spiritual support at a time when it was never more needed. The social worker may have assisted the patient in working through issues with other family members that had gone unresolved for years. The volunteer may have become a valued family friend. The bereavement counselor may have become involved with members of the family even before his death. The emotional, spiritual, and psychological support that the hospice team provides maximize the quality of life for our patients for whatever time they have remaining. It is a disservice to the patient and the family to wait until the patient is barely responsive before allowing the hospice team to begin to assist in caring for the patient.

All insurers in Rhode Island cover hospice care. In 1982, Congress created the hospice Medicare benefit, which allowed terminally ill Medicare beneficiaries access to hospice services. The Medicare benefit provides additional benefits, including:

- There is no requirement that the patient be homebound.
- It provides for coverage of medications used for symptom control of the terminal illness.
- It mandates availability of spiritual, emotional, and psychological support as the hospice program is required to provide chaplains, volunteers, and social workers.
- It includes the patient's family in the services provided; in fact, services do not end at the time of death, but continue with bereave-

ment support by the nurses and bereavement counselor after the patient's death.

One barrier, recognized for physicians considering referral to hospice, is the six-month prognosis. Physicians are not asked, however, to guarantee that the patient will die in six months. They are asked to certified that the prognosis is six months or less if the disease proceeds as expected. We anticipate that some patients will die more slowly than expected. Patients are reassessed at regular intervals. If they no longer appear to have the prognosis of six months or less, then discharge from hospice is initiated.

Efforts continue to find ways to improve patient access to hospice; however, physicians continue to play a key role in the dying process. Recognition of the value of this pre-death emotional, spiritual, and psychological support to their patients will assist in this effort.

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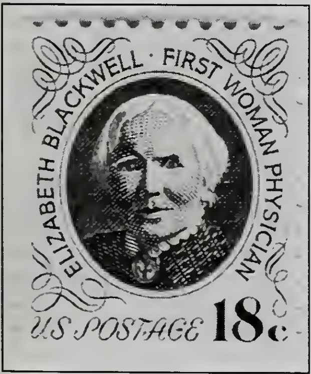
Philately in Medicine

John Tierney

Elizabeth Blackwell

Elizabeth Blackwell, the first woman physician of modern medicine, was born in Bristol, England, in 1821, one of nine children of Samuel and Hannah Blackwell. In 1832 the family emigrated to the United States and lived in New York City for several years before moving to Cincinnati.

Elizabeth decided to study medicine in 1844. When medical schools in Philadelphia and New York rejected her because of her gender, she studied privately with Dr. Samuel Dickson. Finally, she was admitted to the Geneva Medical School, Geneva, New York. In 1849, when she received her MD degree, it made news in the United States and abroad. After graduation, she studied at St. Bartholomew's Hospital in London, then, in 1850, returned to New York City to practice. She encountered prejudice again and opened a private dispensary that became incorporated in 1857 as the New York Infirmary and College for Women, staffed entirely by women.


ELIZABETH BLACKWELL · FIRST WOMAN PHYSICIAN
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In 1869 she settled permanently in England. In 1875 she became a professor of gynecology at the London School of Medicine for Women, a school she had helped to establish. She continued to advance medical training for women in Europe and the United States until 1907, when she was seriously injured in an accident and retired to Hastings, where three years later she died.

In 1949 the Amercian Woman's Medical Association established the Elizabeth Blackwell Medal, awarded to a woman physician for the most outstanding contribution to the cause of women in the field of medicine. In 1974, the United States postal service put her on a United States stamp - the first woman physician so honored.

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NINETY YEARS AGO

[FEBRUARY, 1909]

An editorial urged a school vaccination service, to complement elementary school inspection services. Currently, even when children were offered free treatment, " it was extremely difficult, and in a large proportion of cases impossible, to induce the parents of the children to provide proper medical care..." for conditions (scabies, trachoma, pediculosis, ringworm) detected under "inspection." Consequently, the author urged on-site vaccination, promising that "at present practitioners [will] not lose anything by this municipal medical service."

Key articles focused on the uterus. H.C. Pitts, MD, ("Displacement of the Uterus) discussed the tonic resistance of muscles of the pelvic outlet, and the retentive power of the abdomen. Arthur T. Jones, MD, ("Treatment of Uterine Displacement") advised, as a medical treatment, hot douches, massage tamponade, and keeping a patient in a dorsal position with hips elevated. Surgically, he advised addressing the cause; for example, by removing the tumors or diseased appendages, breaking up adhesions, or letting out pus or blood. He cautioned colleagues to calm the patient: "Her mind should be diverted from her trouble as much as possible."

Harold Palmer, MD, in "Medical Education in Berlin; Advantages Open to American Students," advised students to hire a competent German tutor, and to brace themselves for costly living expenses, unless "one is willing to live just as the natives do, largely on vegetables, wurst, etc."

FIFTY YEARS AGO

[FEBRUARY, 1949]

Two lead articles focused on anoxia of the newborn. William P. Shields, MD, noted that "there are more neonatal deaths than stillbirths," and that "the mortality in the first 15 minutes of life exceeds that of any subsequent month." Furthermore, surviving babies often suffered permanent central nervous system damage. Dr. Shields cited studies at Emma Pendleton Bradley Home that linked childhood behaviour disturbances to cerebral anoxia. Walter Dufresne, MD, urged prevention: physicians should treat and recognize the mother's anemias, cardiac disease, diabetes, symptoms of pre-eclampsia; physicians should brief patients on the danger points; and, during labor, physicians should monitor the heart rate every 30 minutes during the first stage, every few minutes during delivery. He urged caution with medication: "In this modern age, it would be professional suicide to refuse amelioration of pain to the laboring patients, but, in the interest of the unborn a plan must be advanced for a reasonable and judicious use of such medications."

An editorial on "Smoke Pollution" lamented the pollution from coal burners ("We can go outdoors now and rub black oil off the sides of our buildings."), but expressed skepticism about newer fuel ("As we walk down the north side of College Hill and find the Athenaeum obscured by judicial smog we must not be too optimistic about new oil burners.")

TWENTY FIVE YEARS AGO

[FEBRUARY, 1974]

May 17, 1973, to celebrate the opening of the Ambulatory Patient Center at Rhode Island Hospital, Kerr L. White, MD, traced the 300 year-old history of hospital-based ambulatory care. The Journal reprinted his talk ("Ambulatory Patient Care: Epidemiology and Concept of Risk are Shifting Focus of Medical Care from the Hospital Ward to Outpatient Center").

Mary Lekas, MD, FACS, described "Teflon Injection for Vocal Cord Rehabiltiation." She noted that the procedure alleviates aspiration and dysphonia from vocal cord paralysis. In 1911, Bruenings had injected paraffin into vocal cords for correction of laryngeal paralysis, but the improvement was temporary, since paraffin was absorbed. Also, paraffin had a carcinogenic effect. Teflon, though, which was recently approved by the FDA for general usage, is inert and appears safe.

The editorial discussed the dilemma of DDT - banned in the United States, largely as a result of Rachel Carson's *The Silent Spring*, yet admittedly effective against insects. As other countries consider banning DDT, a World Health Organization official warned that "immediate discontinuation of the use of DDT would be a disaster to world health."

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Substance Abuse Treatment in Rhode Island

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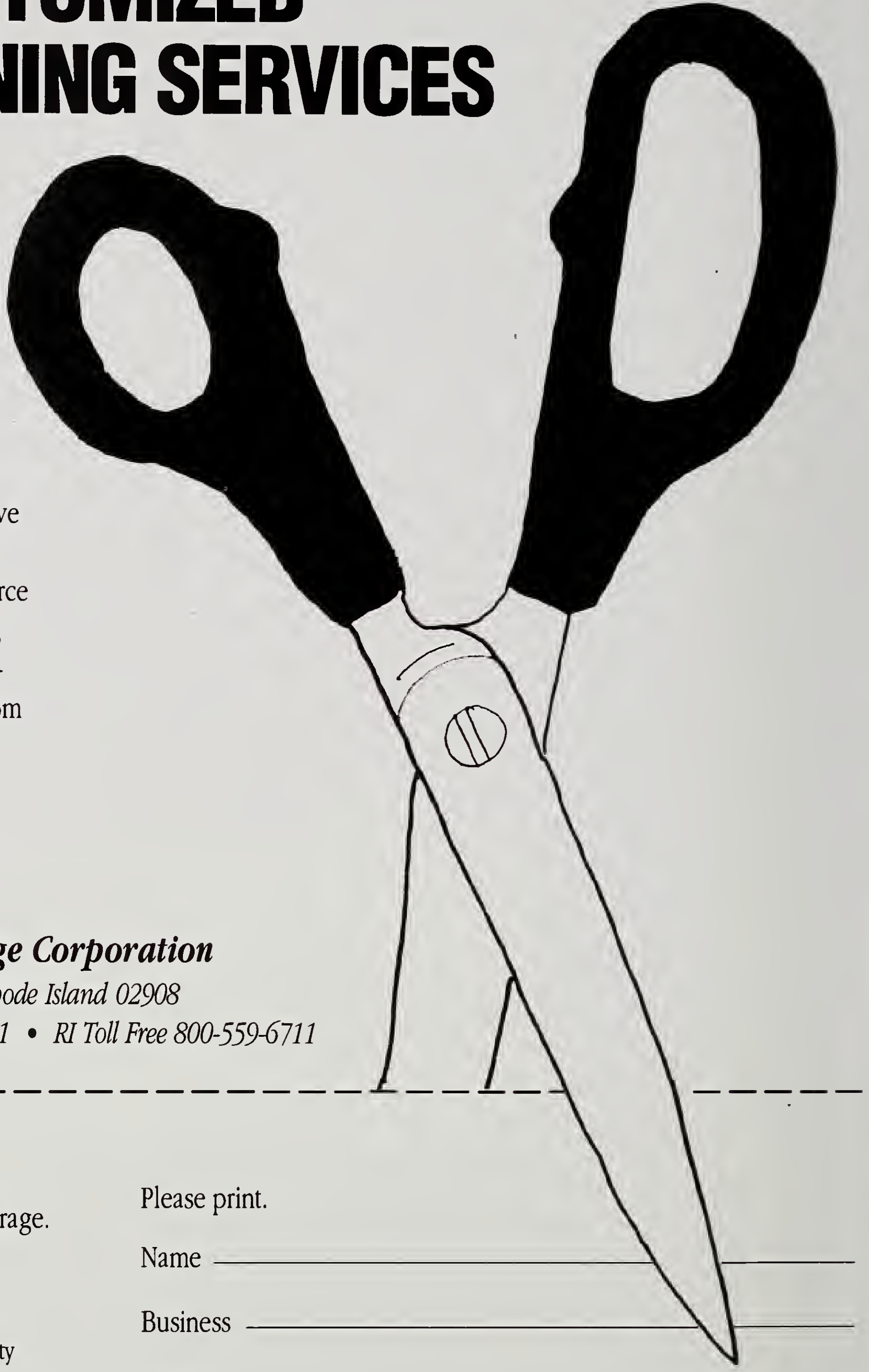

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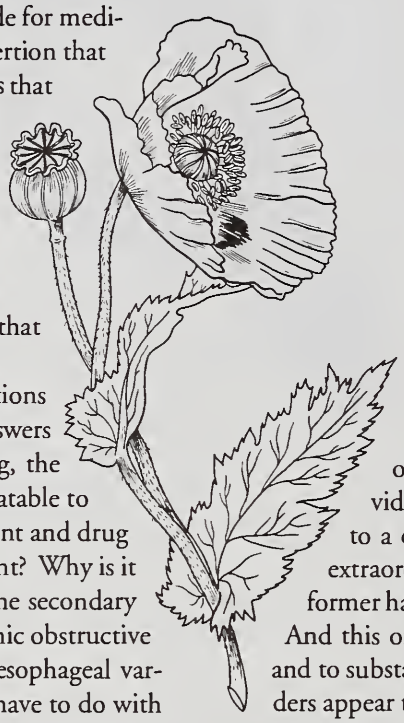
COMMENTARIES

Substance Abuse Treatment: Time for a National Outcome Oriented Approach



In this issue compelling arguments are made for medical treatment of substance abuse. The assertion that treatment efficacy for substance abuse exceeds that for other chronic diseases such as asthma, hypertension and diabetes is made and referenced. Why then is funding so scant and treatment so hard to arrange? Why has the mayor of New York recently announced his intention to abandon methadone maintenance, labeling it as a substitute addiction that masks rather than treats the problem?

This issue does not address these questions because they are not "medical," but their answers are crucial to our waging, let alone winning, the war on substance abuse. Why is it more palatable to spend large amounts of money on punishment and drug interdiction than on prevention and treatment? Why is it easier to get health insurance coverage for the secondary effects of substance abuse (lung cancer, chronic obstructive pulmonary disease, hepatitis, endocarditis, esophageal varies, etc.) than the abuse itself? The answers have to do with our uneasiness with the overlapping concepts of "disease" and character flaw, especially when the abuse is either mind-altering or threatening to societal norms. Does President Clinton have a "sex addiction" or a personality defect? Does an overweight person suffer from the sin of gluttony or an "eating disorder?"

THE POPPY

The opium poppy (*Papaver somniferum*) was reportedly first cultivated for seed in neolithic times. Its oldest known recorded use for medicinal uses occured in Mesopotamia in 2000 BC, though historians suspect the Sumerians used the plant in 5000 BC. Ancient Europe was well aware of the narcotic properties of *P. somniferum* and used the stems, leaves and fruits as a sleeping aid and pain killer. The Greeks are credited with mastering the technique of extracting opium resin by making longitudinal cuts along the capsule, collecting the latex, and chemcially refining the drug. By 1000 AD, the growing of opium was widespread in Europe and Asia. It later became a major trade commodity of the Arab Empire, China and India

– Gretchen Halpert

Reference: *Poppies*. Christopher Grey-Wilson. B.T. Batsford, LTD, London, 1973.

The economics of substance abuse treatment parallels that of mental health. There is something bothersome to the body politic about the notion of a disease without an abnormal laboratory test. We will provide coverage of anorexia nervosa, but only up to a certain point, yet there's no ceiling on the extraordinary costs of treating cancers, despite the former having a better prognosis in younger patients. And this of course extends to other mental disorders and to substance abuse. Our approaches to these disorders appear to fall into one of three categories: the puritanical, which, like the New York City approach, may border on vindictive; the hopeless (especially for substance abuse); and the rational. This issue is a strong endorsement for the rational: a cost-effective, humane set of programs that not only treat but measure outcomes in order to develop better, more efficient treatments in the future.

– Joseph H. Friedman, MD

Science, Policy, and the Future of Substance Abuse Treatment in Rhode Island

Abbreviations Used:

ASAM PPC-2	American Society of Addiction Medicine Patient Placement Criteria, Second Edition
ASI	Addiction Severity Index
FFY	federal fiscal year
SAMHSA	Substance Abuse and Mental Health Services Administration

Abuse of alcohol and other drugs remains one of our country's most intractable and expensive problems. The nation's costs for alcohol and drug abuse are $276.3 billion,[1] which far exceed that of cancer, respiratory diseases, or coronary heart disease.[2] Alcohol and drug abuse in adults lead to lost productivity as well as higher costs for crime, social welfare, and medical/behavioral health disorders. Adolescents and children who use alcohol and other drugs have increased drop-out rates, school absenteeism, tobacco use, emotional and conduct disorders, and learning problems.

Although these problems seem staggering, treatment strategies for drug/alcohol addictions are effective. The National Treatment Improvement Evaluation Study[3] and the Drug Abuse Treatment Outcome Study[4] showed that substance abuse treatment significantly decreased alcohol/drug use, medical visits, psychological distress, homelessness, and crime. Unfortunately, almost 80% of addicted individuals never receive treatment.[5] Substance abuse professionals have not communicated successfully about the cost-effectiveness of treatment. The payers, including health insurers, government, and employers, remain skeptical. In addition, the field struggles with how to assure the use of research-based treatment strategies. Treatment approaches which are effective receive limited attention and use.[6]

For many years, leaders in the field of addictions have stated that we need to measure the outcomes of treatment, for two reasons. First, it provides scientific evidence of the value of treatment and drives public policy, insurance practices, and public perceptions about recovery for addicted persons. Second, it encourages use of the most effective models of treatment. Despite these admonitions, resistance to outcome measurement persists.

There are many reasons for this resistance. Providers worry that outcome measures capture only part of the results of treatment. If providers cannot assure success, they fear being forced out of business although no medical treatment guarantees successful outcomes all the time. In fact, as David Lewis points out, the success of substance abuse treatment compares favorably to that for diabetes, asthma, and hypertension.

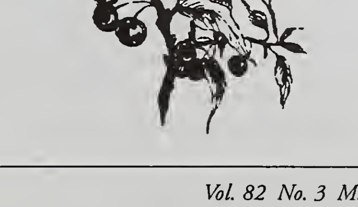
Rising costs, though, are pushing the field to recognize the importance of outcomes measurements. Insurers now look closely at the cost-effectiveness of treatment. If a procedure doesn't reduce overall costs through decreased hospitalization and physician visits, insurers may discontinue coverage. In addition, some federal initiatives drive current practices. The 1994 Government Performance and Results Act mandates that all federal agencies set and meet performance and outcome standards. For states, local governments and private organizations, this mandate has far-reaching implications. Rhode Island will receive $5.9 million from the federal government in FFY1999 to support substance abuse treatment and prevention services. The federal agencies which distribute these funds will require proof of the outcomes.

Traditionally, the major outcome measured in substance abuse treatment has been abstinence. We now understand that the most important outcomes relate to the person's quality of life and impact on society: whether the addicted person is able to obtain and keep employment, avoid public assistance, maintain a safe and secure family environment, lead a crime-free and violence-free life, and not suffer from mental/emotional disorders.[7] For adolescents, we also need to know whether they stay in school and progress satisfactorily. This is where measurement technologies can help.

There are many different outcomes instruments. The Addiction Severity Index (ASI)[8] assesses lifetime and current functioning for adults in seven key areas: medical, employment, alcohol, drug, family/social, legal, and psychiatric. Adaptations of the ASI for adolescents also assess school performance. In 1996, the Rhode Island Department of Health/DSA piloted the ASI for pregnant and parenting women receiving publicly-funded substance abuse treatment at admission, discharge and 6 month follow-up. This study also included an adaptation of the ASI for women.[9] Final results will be released in early 1999. Due to promising preliminary results, the DSA applied for federal funds to expand use of the ASI to clients in state-supported treatment. This grant was awarded in October 1998 to the DSA, which has now been moved to the Department of Mental Health, Retardation and Hospitals. Rhode Island is developing a systemic method to document the outcomes of treatment and to use these results to guide public policy, funding decisions, program planning, and to design training for treatment providers. In addition, this federal grant will involve Rhode Island in national efforts to develop standardized outcome measures for all federally supported treatment services.

In September 1998, the Department of Health/DSA, in cooperation with the Addiction Technology Transfer Center at Brown University and the Drug and Alcohol Treatment Association, sponsored a conference, "Outcomes-Based Substance Abuse Treatment: Survival 2000." Presenters included national leaders such as A. Thomas McLellan, PhD, David Lewis, MD, Norman Hoffmann, PhD, Dennis McCarty, PhD, and Camille Barry, PhD, Acting Director of the SAMHSA Center for Substance Abuse Treatment. Participants learned about treatment outcome evaluation strategies, research results, federal and state initiatives, and practical ways to establish outcomes measurement in treatment facilities. Dr. McLellan stressed that: 1) there are effective methods to evaluate the outcomes of treatment; 2) some treatment models are more effective than others; 3) programs which address employment, psychiatric, and family issues obtain 20-50% better outcomes; and 4) the culture of the treatment field changes slowly. New, effective strategies often differ from historical practices.

Outcome measurement technologies represent only one area of change. Standardized patient placement criteria can determine the best level of treatment (outpatient, intensive outpatient/day treatment, residential, inpatient) for a specific person. The practice of the treatment field, however, eschews systematic placement. The type of treatment received is often determined by availability, resources, and referral practices, rather than patient need and appropriateness. Since placement affects



both cost and outcome, the use of standardized placement criteria is needed.

The American Society of Addiction Medicine (ASAM) developed placement criteria in 1991; the latest version is *Patient Placement Criteria for the Treatment of Substance Related Disorders, 2nd edition* (ASAM PPC-2).[10] These criteria are increasingly used by managed care plans, insurers, and government agencies. Rhode Island adopted these criteria for all state and Medicaid-funded substance abuse treatment services in July 1998.

Robert Marshall and Sherry Knapp start off this issue with a description of the substance abuse treatment and prevention system in Rhode Island and provide a context for thinking about the new technologies, policies, and clinical practices. Other articles highlight treatment outcomes research (Zywiak, Hoffmann and Floyd), pharmacological and behavioral treatment of opiate abuse (Wartenberg), prevention of substance abuse (Dube), and the abuse of drugs/alcohol by physicians (Hamolsky). In addition, David Lewis discusses exciting initiatives to mobilize physician leaders around a new approach to national drug policy.

We hope you will find this special issue informative and enlightening. Substance abuse occurs in 7-12% of your adult and adolescent patients. The approaches and technologies addressed in this issue demonstrate that the ravages of this disease need not go untreated.

– Sherry Knapp, PhD,
and Robert J. Marshall Jr, PhD

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Absinthe Makes the Heart Grow Fonder

When seeking relief from intestinal infestation by worms, primitive peoples often resorted to empirically chosen preparations derived from botanicals. Through random testing, many had selected a particular shrub with inconspicuous blossoms made unique by the extreme bitterness of its leaves. They crushed these leaves and brewed the mash in boiling water to yield an intensely bitter, oily decoction. The Greeks called it *apsinthios,* meaning without sweetness. Beyond its medicinal value, it symbolized to them grief and bitterness. The common name for the herb, reflecting its use, was wormwood.

The Scriptures mention wormwood frequently but almost always in a metaphoric sense and symbolic, again, of bitter sadness and calamity. "To recall my distress and misery was wormwood," says Lamentations [3:19.]

Botanists later called the shrub, *Artemisia,* part of a family of wild perennials that includes sagebrush, tarragon and wormwood. [Artemis, twin sister to Apollo, was a Greek agricultural diety, the wild virgin huntress and, like her brother, a fearsome archer.]

The Greeks sometimes flavored their wines with wormwood extract; and the Romans gave it to their athletes in the fervent hope that it might enhance their strength. Pliny and Dioscorides both describe wormwood as a medicinal herb for the treatment of worms and as an insect repellant.

Artemisia shrubs grow readily throughout Europe and the Middle East, particularly in dry soil. The extracted herbal oils, its intense bitterness taken as proof of its therapeutic effectiveness, were employed during the Middle Ages for many ailments. It also found its way as a flavoring agent of wine-based spirits. Wormwood, in German, became *wermut,* and eventually in English, vermouth, a white wine accented by herbs such as wormwood, anise and coriander.

During the reign of Louis Philippe, the French extended their colonial empire to incorporate Algeria. French troops became enamored of a local beverage, heavily laced with wormwood extract, and said to suppress fevers such as malaria. They brought the drink back with them to France where it gradually assumed considerable popularity. A vintner named Henri Louis Pernod adapted an industrial process to standardize and mass produce this wormwood decoction, calling it now by its older Greek name, absinthe. His process involved the extraction of essential oils from large batches of wormwood, anise, fennel, cardamom, coriander and angelica using grain alcohol as the diluent; this was followed by a distillation step which increased both the alcohol and wormwood concentrations of the final product. The luminous green color of pure absinthe derives from its high chlorophyll content.

Absinthe became hugely popular. An 1894 description of a Parisian street: "... the noon hour is a little fete when people try to forget that they are working for their living. They do not go immediately to eating. They sit outside upon the sidewalk, even in the winter time, look at passers-by and sip their drink. The drink is absinthe. They drink it very slowly; by slow degrees they feel their poor, tired backbones strengthen and their brains grow clearer, and they feel a touch of happiness."

By 1913, the French were tippling 10,500,000 gallons of absinthe per year. It became part of a daily ritual called the green hour [*l'heure verte*]. French laborers, leaving their sites of work, habitually visited the local bar before eventually returning home. Their ritual, observed with great solemnity, consisted of pouring a small amount of emerald-green absinthe into a glass, placing a perforated spoon filled with sugar on the edges of the glass and then slowly pouring cold water through the sugar to produce a cloudy yellowish white fluid with diminished bitterness. Absinthe drinkers reached a dazed, intoxicated state much more rapidly than those who drank wine or spirits alone.

Absinthe spread to Victorian England vying with laudanum as the nation's most popular mind-altering drug.

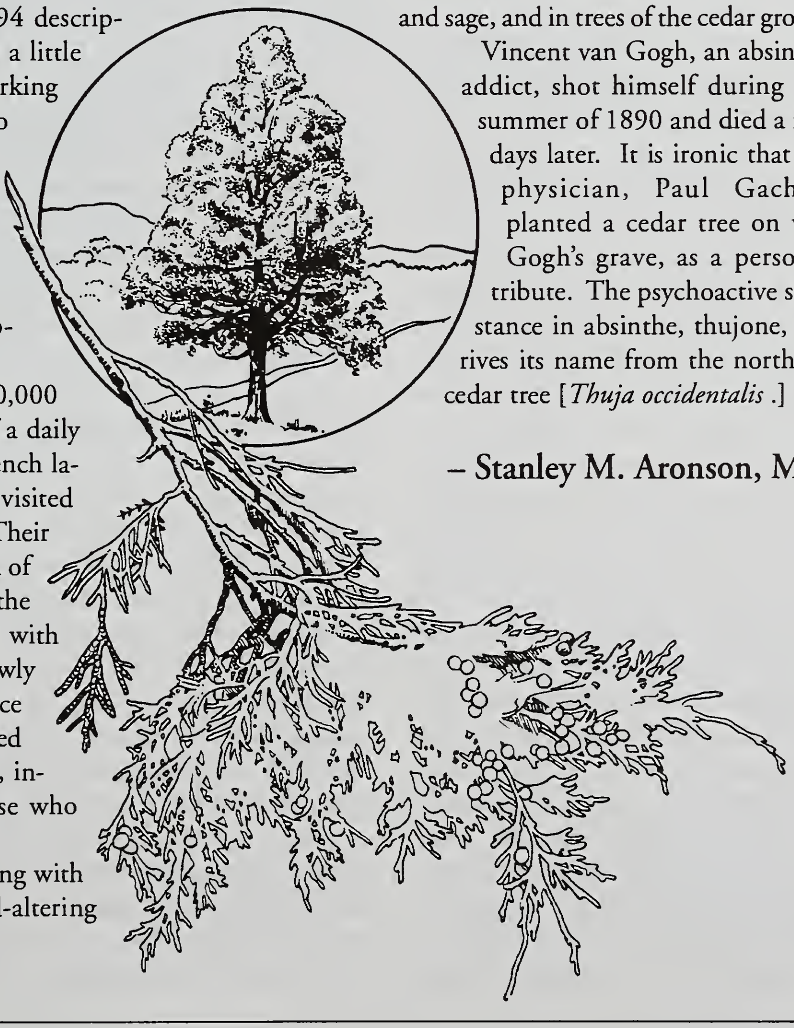
Absinthe drinking achieved immense acceptance, even a sense of close identity, with poets and artists resident in France. Manet, Degas, Toulouse-Lautrec, van Gogh, even Picasso, immortalized the absinthe drinker in a special genre of paintings which faithfully captured the benumbed faces of absinthe addicts. And poets such as Baudelaire and Verlaine claimed that their genius was little more than the liberation, through absinthe, of their latent creativity. By the turn of the century absinthe was heralded as the quickest path to artistic fulfillment.

The unpoetic physicians of France, however, portrayed absinthe with a different face. They documented a drug which regularly caused hallucinations and other psychotic reactions as well as convulsions; and in chronic drinkers, chemical dependency, debilitation and irreversible brain damage. By 1905 Belgium had banned the manufacture and sale of absinthe. Switzerland, Italy and the United States followed within a few years; and France reluctantly prohibited absinthe by 1915. A few European countries still allow its sale. Pernod survives in France today as a popular aperitif but without any significant wormwood content.

Chemists have isolated a toxic substance, called thujone, which appears to be the major hallucinogen in wormwood. In experimental animals it causes the many neurological changes ascribed to absinthe. The addictive nature of absinthe, however, [beyond the obvious role of its diluent, alcohol] has not been fully explained. Thujone, in much lesser concentrations, is also found in certain herbs such as tansy and sage, and in trees of the cedar group.

Vincent van Gogh, an absinthe addict, shot himself during the summer of 1890 and died a few days later. It is ironic that his physician, Paul Gachet, planted a cedar tree on van Gogh's grave, as a personal tribute. The psychoactive substance in absinthe, thujone, derives its name from the northern cedar tree [*Thuja occidentalis* .]

– Stanley M. Aronson, MD



The Substance Abuse Treatment System in Rhode Island: An Ecological View

Robert J. Marshall Jr., PhD, and Sherry Knapp, PhD

Abbreviations Used:

AOD	alcohol and other drugs
ASAS	Adolescent Substance Abuse Survey
ASSIST	American Stop Smoking for Intervention Study
CCRI	Community College of Rhode Island
CIS	client information system
CMHCs	community mental health centers
DCYF	Department of Children Youth and Families
IDU	injection drug use
LCDCS	licensed chemical dependency clinical supervisors
LCDP	licensed chemical dependency professionals
SAPT	Substance Abuse Prevention and Treatment
TASC	Treatment Alternatives to Street Crimes

Substance abuse is both deadly and expensive. Drug and alcohol abuse accounts for up to 500 deaths and more than $600 million each year in Rhode Island. Nicotine addiction, primarily cigarette use, kills even more Rhode Islanders (about 2000 annually) and costs the state about $300 million per year. Taken together, the use and abuse of these substances present a challenge to the social, economic and health care systems of Rhode Island. Practicing physicians play a key role in meeting this challenge through prevention, treatment and policy development.

This article focuses on alcohol and other drug (AOD) abuse. It offers physicians an ecological view of the landscape for substance abuse treatment in Rhode Island— summarizing the extent of the problem and outlining the configuration of services for prevention and treatment.

A BRIEF HISTORY OF SUBSTANCE ABUSE[1]

Compared to other areas of public health, the emphasis on the treatment of substance abuse occurred only recently. Treatment evolved from confinement in jails and almshouses at the turn of the century to medical treatment, "therapeutic communities," detoxification facilities and methadone treatment programs in the 1960s. Options expanded to include both drug-free and chemotherapeutic programs in hospital-based, community residential and ambulatory settings. Many of the programs in Rhode Island today emanate from these local community endeavors.

Cocaine and marijuana use burst through the barriers between lower and upper socioeconomic groups in the 1970s. Use of illicit drugs began, for the first time, to involve significant portions of the working and professional population - creating pressure for health insurers. Rhode Island passed laws requiring private insurance coverage of AOD treatment during the 1980s, and added AOD treatment to the "rehabilitation" services covered by Medicaid. Among its other beneficial consequences, this change focused attention on coordinating health and substance abuse treatment for low-income pregnant women and mothers of dependent children.

The prevention of substance abuse also started more recently than in other areas of public health. Efforts during the 1970s focused primarily on school-based education. By the 1990s, Rhode Island established school health education, community prevention and student assistance programs in many cities and towns.

PREVALENCE OF ALCOHOL AND DRUG USE

Despite these efforts in prevention and treatment, substance abuse persists as a major public health problem. Rhode Island data from recent general adult population surveys are not yet available. However, the 1996 National Household Survey on Drug Abuse[2] estimated that 13 million Americans (6.1% of the US population 12 years and older) use illicit drugs. Marijuana continues as the drug of choice (54% of users) compared with 23% for other drugs. Most illicit drug users in the US are white (74%), followed by African Americans (14%) and Hispanics (8%). Men (8.1%) are twice as likely as women to use drugs. Education correlates negatively with use. The 1995 Rhode Island Adolescent Substance Abuse Survey (ASAS), shows a 7% increase in current (past month) marijuana use among school-aged children between 1993 and 1995[3]. Peer disapproval of marijuana declined by nearly 25%.

Injection drug use (IDU) increases public health concerns. Injecting drug users (and their sexual partners) risk contracting HIV and AIDS, as well as tuberculosis and hepatitis. IDU accounts for the single highest proportion of reported HIV seropositive test results in Rhode Island (39%).[4] In Rhode Island, 30-33% of the primary drug caseload reported IDU.[5]

Alcohol abuse contributes significantly to the substance abuse problem. Nearly 109 million Americans (51% of the US population age 12 and over) use alcohol regularly. About 32 million persons (15.5% of the population) engaged in binge drinking, defined as five or more drinks at a time. About 11 million Americans (5.4% of the population) drink "heavily," posing

Table 1 – Licensed RI Substance Abuse Facilities, number and capacity, 1998

Program Type*	Number of Facilities	Capacity
Detoxification	2	62
Residential Rehabilitation	1	49
Extended Residential (non-medical)	14	323
Female	3	36
Male	5	88
Co-ed	2	125
Adolescent	3	44
Long Term Care	1	30
Outpatient Drug Free	30	Not limited
Narcotic Treatment/Detoxification	8	Not limited
TOTAL	**55**	**434****

* No separate licenses given for Social Setting Detoxification and Outpatient Detoxification Services
**Excludes outpatient programs (drug-free, narcotic treatment)
Source: RI Department of Health, Facilities Regulation

utilization and economic repercussions for the health and specialized substance abuse treatment facilities throughout the country.

ALCOHOL AND DRUG TREATMENT PROGRAMS IN RHODE ISLAND

In order to meet these needs in Rhode Island, a network of substance abuse treatment facilities evolved over the past few decades. As of January 1998, fifty-five licensed substance abuse treatment facilities operated in Rhode Island. Certified specialty programs provide 40 additional services for a total of 90 substance abuse treatment programs over several levels of care, with a total estimated residential treatment capacity of 434 persons. (Table 1). This does not include the capacity of outpatient programs (services not requiring an overnight stay) which is not generally limited by regulation. Length of stay differs widely by facility and program.

The State regulates these facilities and establishes minimum standards for quality, safety and other concerns. Facilities need not have a separate license for each program or satellite facility. Additional sub-categories of services operate under residential, outpatient and narcotic treatment facility licenses. Hospitals may provide substance abuse treatment services without a separate license or certification. Of the eight Community Mental Health Centers (CMHCs) in Rhode Island, seven also hold substance abuse facility licenses and provide treatment.

SUBSTANCE ABUSE TREATMENT PROFESSIONALS

Many health professionals treat substance abusers in Rhode Island. Treatment falls within the general scope of practice for more than 25,000 physicians, nurses, psychologists, social workers, mental health counselors, and marriage and family therapists in Rhode Island. Many professional societies offer certification for specialized training and experience in substance abuse treatment. There are also 219 (January 1998) Licensed Chemical Dependency Professionals (LCDP) and Licensed Chemical Dependency Clinical Supervisors (LCDCS) in Rhode Island. However, an estimate of the total number of health professionals regularly in all fields who regularly treat substance abuse is not available.

Getting insurers to pay for substance abuse treatment involves considerations other than licensing. Health plans typically establish networks of substance abuse providers. Often, these are "carved out" or separately managed from networks of physical health care. Most health plans impose provider and facility credentialling requirements. For specialized substance abuse treatment other than by a physician or other doctoral-level provider, minimum credentials often require a Master's degree and 5 years experience.

Effective treatment of substance abuse requires specialized training. Several local academic institutions offer relevant coursework: the University of Rhode Island, Roger Williams University, Community College of Rhode Island (CCRI), New England School of Addiction Studies (Maine) and Brown University (Addictions Technology Transfer Center of New England).

ADMISSIONS TO SUBSTANCE ABUSE TREATMENT FACILITIES IN RHODE ISLAND

Information on admissions to substance abuse treatment facilities comes from several sources. The state's Client Information System (CIS)[5] monitors admissions to publicly-funded community detoxification and treatment providers. This system excludes a few privately-funded treatment centers, hospital admissions for substance abuse treatment, out-of-state admissions and an unknown number of admissions to private, independent outpatient practices. However, it provides a good estimate of the admissions flow through reporting facilities.

In state fiscal year 1997 (FY97) the CIS recorded 11,972 alcohol and other drug admissions to Rhode Island detoxification and treatment facilities. (Table 2) These represent 8,030 separate individuals served. Multiple admissions reflect recidivism as well as an individual's movement through a continuum of treatment, such as from detoxification to residential therapy to outpatient therapy. Nearly 60% of all FY97 admissions went to ambulatory settings.

Most admissions came from Providence (31%) with Pawtucket (8%); Cranston, (7%); Warwick, (5%); New-

Table 2 - Substance Abuse Admissions by Facility Type, Rhode Island, FY97

Type of Facility	Admissions FY97
Free standing (non-hospital) residential detoxification	3701 (31%)
Residential Treatment (non-hospital) (Short-term = 53; Long-term = 1055)	1108 (9%)
Outpatient Treatment	4728 (39%)
Intensive Outpatient/Day Treatment	230 (2%)
Outpatient Methadone Maintenance and Detoxification	2205 (18%)
TOTAL	**11972 (100%)**

Source: RI Department of Health, Substance Abuse

port, (3%) far behind. Caucasians represented 76% of all admissions followed by 10% Black, 9% Hispanic/Latino, 1% for each of American Indian and Cape Verdean and 2% all others. More than half cite "public assistance" as the main source of income. Most clients are unemployed or underemployed, and have less than high school education.

Admissions reflected 70% male and 30% female clients. Of the female caseload, 93% were between the ages of 12-44 - raising concerns about substance abuse during pregnancy and the impact on children's protective services. Of overall admissions, 7200 (60%) involved primary drug use; 4772 (40%) involved primary alcohol use. Nearly two-thirds of primary drug users used heroine; two-fifths of users injected the drug.

Although some plans are becoming more enlightened, substance abuse provides an easy target for achieving economies as managed care continues to ratchet down the cost of health services.

◆

The CIS data include other kinds of information about admissions, such as referral source and sources of payment (Figure 1). The Rhode Island network operates primarily by self-referral using publicly-funded treatment administered by the State. Self-referrals constituted the majority (52%) of all admissions to detoxification and treatment programs. Of the remainder, health care providers, mental health programs or other substance abuse treatment providers referred 16%; the Criminal Justice System referred 23%. The Department of Children Youth and Families (DCYF) referred 5%, but less than 1% came from schools, employers and all other sources. Categorical public (state/federal) funds supported 65% of all reported admissions, followed by self-pay (12%), Medicaid and RiteCare (12%) and private insurance (8%). Veterans Administration and other state programs paid for less than 3% of all admissions.

Hospital Admissions for Substance Abuse

Acute care hospitals in Rhode do not submit data to the CIS. However, all but two hospitals regularly report discharges to the Department of Health. They reported 1275 discharges related to substance abuse (alcohol and drugs) during FY96. Roger Williams Hospital treated 53%, followed by Rhode Island Hospital (16.5%) and Newport Hospital (8%). These hospitalizations account for 5,531 in-patient days or an average of 4.3 days per discharge. More than half (55.1%) of all substance abuse related hospital discharges are among patients ages 25-44. This differs from other non-maternity hospital discharges, which generally reflect older populations. However, about 25-40% of all US hospitalizations are not diagnosed as substance abuse, but occur as a result of alcohol and other drug abuse. These include injury (unintentional and intentional), cancer, neonatal conditions and other diseases of the liver, pancreas and gastro-intestinal tract which are related to substance abuse.[6]

Figure 1
Sources of Referrals and Payments
Substance Abuse Admissions, FY98

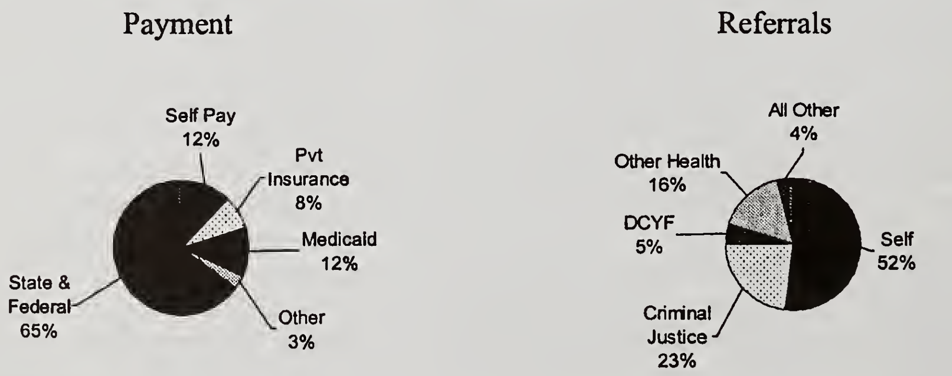
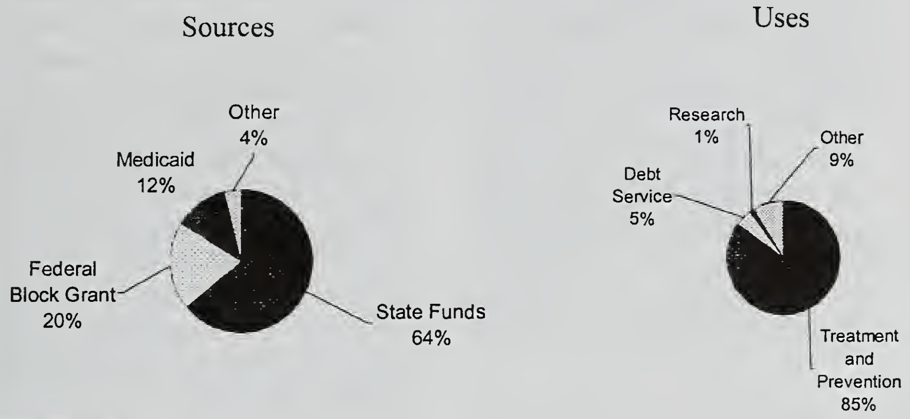


Figure 2
Sources and Uses of Core Public Funds
Substance Abuse, FY98

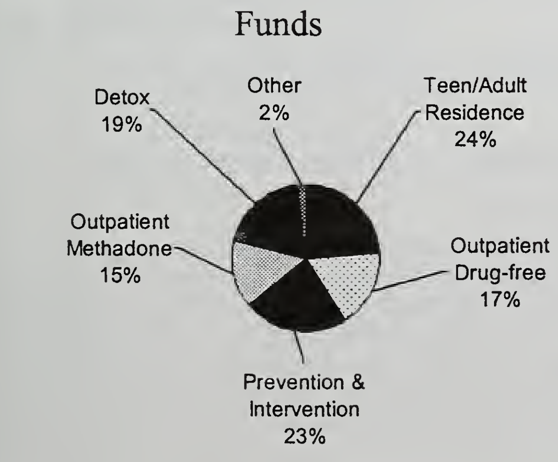


FINANCIAL RESOURCES FOR SUBSTANCE ABUSE TREATMENT IN RHODE ISLAND

Rhode Island uses a complex funding mix for substance abuse treatment. Funding includes: private health insurance, federal categorical (block grant) funds, categorical state appropriations, federal/state medical assistance (Medicaid/RiteCare), self pay and client out-of-pocket payments, private philanthropy, special federal grants and contracts, related state and federal programs (e.g. corrections, child protective services, Veteran's Administration) and other components.

State financial data from FY98 provide some information about sources of funding. Categorical public funding for substance abuse services in Rhode Island during FY98 totaled approximately $20.7 million. This "core funding" included general state revenue funds (64.1%), federal Substance Abuse Prevention and Treatment (SAPT) Block Grant funding (20.0%), Title XIX (Fee-for-service Medicaid) state and federal funds (12.3%), specialized federal categorical grants (3.4%), and restricted receipts (0.2%). Most of these resources are dedicated to the purchase of treatment and prevention services (85.3%). Other amounts went toward debt service on 11 publicly owned facilities which house treatment programs (5.3%), contracts for research projects (0.9%), and all other expenditures (11.5%).

Figure 3
Expenditure of Core Funds
by Treatment Modality, FY98



*Based on $17.7 million in state/federal funds

Figure 3 shows a breakdown of core funding by primary treatment modality. These amounts reflect only those resources available through state-administered treatment programs, not other state, federal or private sources mentioned above. Responsibility for administering state substance abuse programs moved to the Department of Mental Health, Retardation and Hospitals in September 1998.

The State of Rhode Island also supports a small case management staff in the Treatment Alternatives to Street Crimes (TASC) program. These professionals access, refer and manage substance abusers who are diverted by the Courts from possible incarceration into treatment - including individuals cited for offenses for driving while under the influence. The TASC staff handled about 2200 cases per year.

SUBSTANCE ABUSE PREVENTION SERVICES

The public health model of substance abuse addresses the agent (alcohol and drugs), the host (the individual), and the environment (society). This approach opens up opportunities to prevent substance abuse by changing the relationship between two or more components. The main prevention strategies[7] include: Information Dissemination, Education, Alternative activities, Problem Identification and Referral, Community-based Process, and Environmental Initiatives. Programs representing these strategies operate throughout Rhode Island, employing resources from public and private sources. These programs include: 35 municipal prevention task forces; student assistance services in junior and senior high schools; Safe and Drug Free Schools and Communities initiatives; private agency-based programs; a statewide prevention training and resource center and a crisis intervention hotline. The State allocated $4.6 million to community substance abuse prevention activities in FY98. These were supplemented by other prevention funds such as Safe and Drug Free Schools and Communities awards to local schools ($2 million), Governor's Office on Highway Safety funds for injury prevention ($500,000) and federal American Stop Smoking for Intervention Study (ASSIST) resources for smoking prevention ($900,000).

CONCLUSIONS

Rhode Island offers an extensive system of services for prevention and treatment. This system provides a backdrop for the issues and practices discussed elswhere in this issue. These include the role of primary care physicians in detecting and responding to substance abuse in patients, the effective treatment of opioid users, measuring treatment outcomes, and a national drug policy focused on the treatment. There are resources, programs and professional colleagues available to support and enhance every physician's efforts.

However, many challenges remain. A major one involves assuring that access to treatment is commensurate with the need. This requires better estimates of the need for treatment as well as making adequate resources available to support treatment access. The role of private health insurers in providing access to treatment also needs to be resolved. Health plans typically apply unique restrictions and limitations to substance abuse treatment. Although some plans are becoming more enlightened, substance abuse provides an easy target for achieving economies as managed care continues to ratchet down the cost of health services. Finally, there are too many unanswered questions about how the substance abuse sector works. Resolving this will require additional information on the extent and severity of addiction in the population, objective measures about the level of care (placement) and treatment intensity, and indicators of treatment outcome, including employment, housing and other correlates of recovery. Addressing these concerns will move us one step closer to our goal of assuring safe and healthy lives in safe and healthy communities.

Gretchen Kai Halpert

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Enhancing Alcohol Treatment Outcomes Through Aftercare and Self-Help Groups

William H. Zywiak, PhD, Norman G. Hoffmann, PhD, and Anthony S. Floyd, PhD

Abbreviations Used:

AA	Alcoholics Anonymous
HMO	health maintenance organization

Physicians and other health care professionals often encounter individuals dependent upon alcohol. Less widely recognized is that physicians occupy a prime position to assist in stabilizing recovery by encouraging patients to use aftercare and self-help programs. Addicted individuals use health care services more frequently;[1] this facilitates recognition of substance use disorders. Screening can be done with a brief inquiry. Free screening measures are available[2] and even a few items consistently asked can produce reasonable detection[3] (Table 1). Questions about drinking-related problems yield positive screens by avoiding questions about quantity and frequency of drinking.[4]

The vast majority of treatment programs for alcohol dependence use the 12-step model. Naturalistic studies show that most patients benefit from this treatment.[5] Getting alcoholics into treatment should be the prime concern of physicians and third-party payers. Successful treatment of alcohol disorders leads to better health and reduced health care costs for the recovering person,[6] family members,[7] health plans and purchasers of health insurance.

As with any chronic illness, sustaining the gains from treatment requires additional maintenance care and behavioral management of the illness. The literature shows a consistent relationship between continued contact with treatment professionals and better outcomes.[8] Aftercare and self-help group attendance (e.g. AA) following treatment also demonstrate a consistent relationship to better outcomes.[5,9,10] When meeting with patients who have completed substance abuse treatment in the past 6 months, physicians and other health care professionals should determine current participation in aftercare and self-help groups. Attendance in both should be encouraged.

Table 1
The UNCOPE

U	"In the past year, have you ever drank or used drugs more than you meant to?"
N	"Have you ever neglected some of your usual responsibilities because of using alcohol or drugs?"
C	"Have you felt you wanted or needed to cut down on your drinking or drug use in the last year?"
O	"Has your family, a friend, or anyone else told you they objected to your alcohol or drug use?"
P	"Have you ever found yourself preoccupied with wanting to use alcohol or drugs?"
E	"Have you ever used alcohol or drugs to relieve emotional discomfort, such as sadness, anger, or boredom?"

Using all 6 items and a score of 2 or more to indicate any abuse or dependence, sensitivities in a clinical population for alcohol, cocaine, and marijuana are 93%, 94%, and 82% respectively. Specificities for this cut-off are 97%, 99%, and 97% respectively. The use of just the "U" and the "C" items and a cut-off of 1 also provide adequate, although somewhat lower sensitivities and specifities. (Item sources: Brown RL, Leonard T, Saunders LA, et al. A two-item test for alcohol and other drug problems. *J Fam Pract* 1997; 44: 151-160; Hoffmann, NG, Harrison, PA. *SUDDS-IV: Substance Use Disorders Diagnostic Schedule, St.* Paul, MN: New Standards, Inc. 1995.)

This study examines the impact of the amount of substance abuse treatment, amount of aftercare, frequency of AA attendance, clinical risk factors, and demographic risk factors on treatment success. It offers guidance for physicians, particularly regarding the importance of aftercare and participation in AA.

METHODS

This naturalistic, prospective study included 1,725 patients with a current diagnosis of alcohol abuse or dependence. All completed substance abuse treatment in the northeastern United States. The average enrollment in the study across the clinics exceeded 90%. In the twelve months prior to treatment entry, 43% of the subjects drank daily, and 42% used drugs at least monthly. The most frequently used drugs included marijuana (41%), cocaine (33%), and minor tranquilizers (30%). Most (77%) of the sample received inpatient treatment only. The majority of the remaining subjects received evening outpatient treatment only. So that the length of treatment across substance abuse treatment programs could be compared using a single

Table 2
Sample Description
N = 1725

Age: mean = 37.9 years standard deviation = 11.1 years
Education: mean = 12.7 years standard deviation = 3.2 years
Percent Male = 78%
Percent Caucasian = 90%
Percent African American = 7%
Percent Married / Cohabiting = 46%
Working Full-Time = 70%

metric, researchers counted each day of inpatient treatment as 5 hours of outpatient care. Only clients completing at least 20 "hours" of care participated in this study. Treatment, thus defined, averaged 109 hours (sd = 41 hours) or 21.8 days. During the six months following treatment discharge, the sample received an average of 2.9 months (sd = 2.5 months) of aftercare. Most (68%) of the sample attended AA at least weekly. Table 2 reports other characteristics of the sample.

Treatment staff collected demographic and clinical information during intake. They recorded the total amount of services provided upon discharge. Phone interviewers employed by an independent evaluation company collected follow-up information at 6 and 12 months post-discharge. The 6-month post- discharge interview included questions about the number of months of aftercare received and the frequency of AA attendance (never, stopped going, once a month or less, several times a month, once a week or more) during the 6 months following treatment discharge. Researchers defined treatment outcome in two ways. First, researchers tracked the number of months of any alcohol or drug use during months 7 through 12 following treatment discharge. (Seventy-two percent of the sample abstained completely; 11% used a substance during all 6 months). Second, researchers dichotomized the outcome variable into "completely abstinent" or "having any relapse during months 7 through 12." This allows an evaluation of the subsequent impact of AA attendance and aftercare during the initial 6 months following treatment. This study also examined two previously developed risk factors (Table 3), clinical risk factors (e.g., current cocaine use) and demographic risk factors (e.g., unemployed, no high school degree).

RESULTS

Multiple regression analysis revealed that five predictor variables were significantly related to the number of months of any alcohol or drug use during the second 6 months after treatment: the amount of primary substance abuse treatment, the amount of aftercare, the frequency of AA attendance, the clinical risk index, and the demographic risk index.

Researchers did not manipulate treatment parameters (e.g. amount of substance abuse treatment and aftercare). Thus we expected, and found, that the amount of substance abuse treatment correlated with clinical risk ($r = .13$, $p < .001$). However, the amount of substance abuse treatment correlated negligibly with the demographic risk index ($r = -.06$, p = n.s.). This suggests that treatment programs take into account clinical severity, *but not demographic risk factors when determining the appropriate amount of treatment.* Researchers observed no significant correlations between the clinical risk index and either aftercare or the frequency of AA attendance. In the case of the demographic risk index, the correlation with aftercare was also not significant. But the analysis produced a small, significant negative correlation ($r = -.12$, $p < .001$) with AA attendance. This suggests that some of the individuals with higher demographic risk were less likely to attend AA. The amount of substance abuse treatment was not significantly

Table 3
Clinical and Demographic Risk Factors: Definitions and Distributions

Clinical Risk Factors

1. current cocaine abuse or dependence diagnosis
2. current marijuana abuse or dependence diagnosis
3. monthly use of another drug (besides alcohol) in previous 12 months
4. use of three or more drugs in week prior to admission
5. any intravenous drug use ever
6. substance abuse treatment in the past 2 years
7. 5 or more antisocial behaviors prior to age 15

Demographic Risk Factors

1. less than 25 years old
2. no high school diploma or General Equivalency Diploma
3. no 4 year college degree
4. unemployed (and not a homemaker or not working by choice)
5. never married

Clinical Risk Factor Distribution:

value	%
0	47%
1	21%
2	13%
3	9%
4	6%
5	3%
6	1%
7	< 1%

Demographic Risk Factor Distribution

value	%
0	12%
1	47%
2	27%
3	11%
4	3%
5	< 1%

Table 4
Abstinence Rates for Months 7-12 After Treatment Depending on Demographic Risk and Length of Substance Abuse Treatment

Demographic Risk and Treatment	% of Sample	% Abstinent
Low Demographic Risk		
Less than 35 hours of Treatment	7%	57%
35 hours or more of Treatment	79%	77%
$X^2(1,N=1481) = 22.3, p < .00001$		
High Demographic Risk		
Less than 75 hours of Treatment	3%	26%
75 hours or more of Treatment	11%	64%
$X^2(1,N=244) = 23.9, p < .00001$		

correlated with amount of aftercare. However, those who received more substance abuse treatment more likely attended AA on a regular basis ($r = .11$, $p < .001$).

Recursive modeling[11,12] identified cut-off points in the five predictor variables used to identify simple rules to optimize treatment outcome. In this case, researchers dichotomized treatment outcome into "completely abstinent" versus "any relapse during months 7 through 12" following treatment completion. Of the two risk factors, the demographic risk index produced the greatest differentials. Fourteen percent of the sample scored 3 or more on the scale and had an abstinence rate of just 56% during months 7 through 12. This contrasted with 75% abstinence for those with fewer demographic risk factors. Patients with two or more clinical risk indicators abstained 68% of the time compared to 74% of those with fewer risk factors. Clearly, the risk factors are related to outcomes. Viewed from the perspective of a clinician, these risk factors also indicate which cases may require closer attention.

Substance abuse treatment achieved the best outcomes when patients received at least 35 "hours" of care. The few (9%) patients who received less than 35 hours of care had only a 50% probability of being abstinent during months 7 through 12 - compared to the 74% probability for those receiving more treatment. Successful recovery requires a sufficient initial dose of treatment services.

> *Getting alcoholics into treatment should be the prime concern of physicians and third-party payers. Successful treatment of alcohol disorders leads to better health and reduced health care costs for the recovering person, family members, health plans and purchasers of health insurance.*

AA attendance produced more striking differentials. Of those who attended AA meetings at least several times a month, almost 80% were abstinent in the second 6 months, compared to 52% for those who attend AA less frequently or not at all. Aftercare utilization proved similar: Those who failed to attend at least two months of aftercare reported an abstinence rate of 62%; those with 2 to 5 months of aftercare reported 75% abstinence; those with 6 months of aftercare reported 82% abstinence.

Next researchers split the sample using the clinical risk index and determined the cut-point for length of treatment. For the low clinical risk group the cut-point occurred at 35 hours. For the high clinical risk group, no discernible cut-point emerged. This indicates that longer substance abuse treatment produces improved outcome for this group, but no clear cut-off optimizes the outcomes. However, the demographic risk index showed clear cut-points in both groups. These two cut-points differed. For the low demographic risk group the cut-point occurred at 35 hours. For the high demographic risk group, the cut-point doubled to 75 hours. High demographic risk patients receiving less than 75 hours of care reported a 26% chance of abstinence. Those receiving at least 75 hours of care increased this rate to 64% (Table 4).

Finally, researchers considered aftercare and AA attendance in combination. The 16% of the sample who

Table 5
Abstinence Rates for Months 7-12 After Treatment Given AA Attendance and Aftercare Utilization

AA and Aftercare	% of Sample	% Abstinent
AA Non-attenders		
Less than 2 months of aftercare	16%	46%
2 to 6 months of aftercare	11%	62%
$X^2(1,N=474) = 11.0, p < .001$		
AA Regular Attenders		
less than 2 months of aftercare	23%	73%
2 to 5 months of aftercare	22%	80%
6 months of aftercare	28%	85%
$X^2(1,N=1251) = 18.1, p < .0001$		

did not attend AA regularly nor attend a minimum of two months of aftercare realized only a 46% abstinence rate. Those who received at least two months of aftercare but did not attend AA regularly, achieved a 62% abstinence rate. Those without the minimum of two months of aftercare, but with regular AA attendance reported a 73% abstinence rate. The study found the biggest gains among those who both attended AA on a regular basis and received the full 6 months of aftercare. They achieved an 85% abstinence rate during the second 6 month follow-up period after treatment (Table 5). Clearly treatment success improves with AA meeting attendance and longer aftercare.

DISCUSSION

Treatment produces positive results for the majority of individuals — provided they receive an adequate amount of substance abuse treatment, have a sufficient duration of aftercare, and use self-help groups. While current treatment addressed clinical risk (e.g. with more treatment), this does not adequately address demographic risk. High demographic risk patients require more attention. Those receiving less than 75 hours of substance abuse treatment demonstrate especially poor outcomes. Findings regarding different cut-offs for length of treatment depending on the demographic risk indicate that a single optimal length of treatment cannot be adopted. Patient characteristics need to be considered when determining the appropriate length of substance abuse treatment.

Overall, more aftercare and more frequent AA attendance produce better outcomes. If physicians and primary care providers advocate for aftercare and self-help groups among patients recently completing substance abuse treatment, abstinence is more likely.

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Treatment Considerations in the Opioid Dependent Patient

Alan A. Wartenberg, MD

Opioid dependency is a major individual and public health problem in the United States, resulting in serious morbidity and mortality, as well as family and societal costs. The U.S. epicenter of heroin use is the Northeast, with more than half the cases in the New York City area, with additional large numbers found in metropolitan areas between Washington, DC, and Boston, including those in Rhode Island. While most opioid use involves injection, substantial numbers may use by nasal insufflation ("snorting" or "sniffing'), and some by smoking. Use of oral fixed-dose combination analgesics containing opioids is widespread and may be associated with toxicity from acetaminophen or aspirin.

Opioid-dependent patients present special challenges to health care. They often have significant underlying psychiatric illness, usually personality disorders, post-traumatic stress disorder and major affective disorders, especially in women. Because of the illegal nature and the high daily cost, users often are part of a criminal subculture, and may present serious management problems in the medical care setting.[1] In addition, drug- seeking behaviors become part of the lifestyle, implicating physicians and other prescribers. Patients who abuse opioids often abuse other drugs, including alcohol, cocaine, benzodiazepines and other sedative-hypnotics. Opioids may be used with cocaine ("speedballs"), or with antihistamines, particularly promethazine (Phenergan®). Drug interactions and other drug effects must always be considered.[2]

Unfortunately, health care professionals receive little training in the management of this common medical problem. While most medical schools include an introduction to the topic, few provide any formal exposure to Addiction Medicine. Likewise, most mentors lack experience in this area. This leads to a self-fulfilling prophecy, in which clinicians feel helpless and hopeless about these patients. Many clinicians are unable to deal competently with the biomedical needs, only increasing the likelihood of behavioral problems, poor outcomes and mutual mistrust.[3] The association between injection opioid use, HIV disease and chronic viral hepatitis leads to greater numbers of these patients entering the health care system. Clinicians must develop skills in the management of these conditions equal to their management of other chronic disorders.

Abbreviations Used:

HMO	health maintenance organization
LAAM	levo-alpha-acetylmethadol
MMTP	methadone maintenance treatment program
OST	opioid substitution therapy
UROD	ultra-rapid opioid detoxification

There have been several recent reviews of the medical complications of opioid abuse[4,5] as well as the management of the opioid abuser in the medical setting.[6,7] The clinician should discuss the course of treatment and begin symptomatic treatment of withdrawal as quickly as possible. A therapeutic contract, either formal or informal, spells out the obligations of the caregiver and the circumstances under which care can be delivered. Clinicians should have an empathetic, nonperjorative and nonjudgmental discussion with the patient on maintaining a safe environment. Patients benefit from instruction on how they can present their needs to staff without displays of anger, threats or drug misuse. When possible, involve all appropriate members of the treatment team, including on-call or covering personnel. This decreases the chance of misunderstanding and staff-splitting behaviors. Patients should be made aware of available options if they feel they are not being treated properly.

Treatment options for opioid dependent patients include short-term detoxification using a number of methods: clonidine, methadone,[6,7] buprenorphine,[8,9], as well as ultrarapid opioid detoxification.[10] The literature provides an excellent recent review.[11] To be successful, detoxification should be followed by structured residential programs or highly structured outpatient programs. These methods suffer from a high relapse rate, with associated morbidity and mortality. Highly motivated patients, such as health professionals, may do well with short-term detoxification and the use of naltrexone as a maintenance drug. For some, complementary methods such as acupuncture may be helpful.[12]

Opioid Substitution Therapy (OST) produces high therapeutic retention, reduced relapse rates and high cost-efficacy. OST usually involves methadone (methadone maintenance treatment programs or MMTPs), but increasingly extends to LAAM (levo-alpha-acetylmethadol) and, in the near future, buprenorphine. While many controversies surround OST, especially in the twelve-step recovery community, patients on these programs demonstrate improved survival, recovery outcomes, improved function and better health than those who do not remain in treatment.[13,14,15] For many patients, the choice is not between methadone/LAAM and abstinence-based recovery, but between OST and relapse to heroin and other street opioids - including the mortality and morbidity associated with relapse and continued use. Methadone, LAAM and buprenorphine treatment will be discussed below.

Ultra-rapid opioid detoxification (UROD) has recently received significant attention.[10] In this technique, the patient receives large doses of opioid antagonists while under general anesthesia, causing rapid receptor reregulation. In theory, the patient can be "detoxified" in as little as four hours. While proponents hail the method as a breakthrough,[16] there have been calls for caution and further study by the Chair of the American Society of Addiction Medicine's clinical efficacy project[17] and by one of the principal investigators of the technique.[18] They recommend UROD only in highly selected patients after informed consent concerning alternatives, risks and benefits. In addition, the routine use of naltrexone (sometimes in the form of implants), following UROD may be associated with overdose risk if relapse occurs.[19]

GENERAL CONSIDERATIONS IN TREATMENT

Appropriate treatment always includes involvement in mutual help programs. These may be twelve-step oriented (Alcoholics Anonymous, Narcotics Anonymous), or newer complementary programs such as SMART recovery (formerly Rational Recovery), Women for Sobriety, Secular Organization for Sobriety and others. In addition, the involvement of family and significant others is critical, both for the health and recovery of family members themselves and the recovery of the affected individual. Family self-help, such as Alanon, Naranon, Families Anonymous, and more formal family individual and group therapies are usually available in treatment settings.

Health professionals involved in the treatment of chemically dependent patients should develop referral resources in their community. These include Addiction Medicine, Addiction Psychiatry, Social Work or others available specialists. Recruitment of chemcial dependency professionals, or training existing staff in chemical dependency issues, may offer programs their own alternatives to referral. Addiction consultation-liaison services may be created. The Conference of Boston Teaching Hospitals organized an effort to develop groups of Internists, Psychiatrists, Pediatricians and Adolescent Medicine specialists, ob/gyn providers, Nurses, Social Workers and others in each institution to provide consultation, training and referral of patients with chemical dependency. The lack of such services in a hospital, HMO, clinic or group practice should be considered a priority for development.

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METHADONE

Methadone produces good results for short-term detoxification (up to 6 months), or as a longer-term OST - particularly in those with longer histories of use or previous failure in residential treatment. Methadone is a synthetic opioid with a long half-life and many active metabolites. Dosing occurs daily. Dosage varies according to region, the experience of the clinical staff and program philosophy. Generally most patients receive between 80-120 mg per day, with some patients doing well at both lower and higher levels. When patients are properly stabilized on methadone, they do not get "high" on it, and function normally.[13,14]

The dose of methadone should be individually titrated. The optimal dose depends on the level of heroin use, individual tolerance, and differences in the individual's metabolism of methadone. The ability to measure peak and trough methadone serum levels has assisted in individualizing dose. It is not uncommon to have patients on 200 mg or more, or receive multiple doses of methadone per day in some MMTPs.[14] Some patients may do well with short courses of treatment— particularly those with a short history of chemical dependency, high levels of motivation and good treatment resources. Patients with longstanding abuse, high involvement in drug lifestyle, and multiple treatments with rapid return to drug use require long-term maintenance, which may be indefinite.[15]

MMTP patients receive regular counseling and random urine toxicology screens. They must comply with clinic rules to remain in treatment. Some patients use illicit drugs and alcohol, or obtain prescription mood-altering drugs while in MMTPs. However, for the majority of patients, drug use and criminal involvement decrease while employment, school and family/other social functions improve. "Harm reduction" provides an important philosophical underpinning to MMTPs, with fewer patients being discharged only because they continue to use. As a result, treatment retention improves and there is a reduction in morbidity and mortality.[13,15]

Patients doing well in treatment may earn "take-home" or "take-out" doses. They need not visit the clinic every day. Such doses are highly regulated, and require compliance with all clinic rules and regulations. After 2-3 years of lack of illicit use, good attendance at clinic and meeting treatment goals, some patients may only return to the MMTP once or twice weekly. Some experts call for allowing such patients to receive prescriptions from specially licensed community physicians as a form of "medical maintenance."

Patients on methadone may be hospitalized for a variety of related and unrelated medical, surgical or psychiatric issues. If hospitalization occcurs, the physician must contact the MMTP to verify the amount and date of the patient's last methadone dose. MMTPs need to know when patients are in the hospital - otherwise they may discharge them due to lack of attendance. If the patient is admitted at a time when the MMTP cannot be contacted, administer the patient's stated dose in stages. For example, a patient receiving 90 mg can get 30 mg every 8 hours. Observe for oversedation, pupillary miosis or other signs of toxicity.

Many patients on methadone now receive doses well in excess of 120 mg per day. This reflects both the higher quality and quantities of available heroin, as well as the ability to measure peak and trough methadone levels and adjust doses accordingly.[14] Upon patients' discharge, inform the MMTPs of the discharge date, last dose received and time of dosing. If other psychoactive drugs are given, the MMTP clinical personnel should be informed, particularly if patients continue to receive outpatient treatment.

Methadone interacts with many drugs. Rifampin, rifabutin, phenytoin, carbamazepine, barbiturates and alcohol increase methadone metabolism and may precipitate withdrawal. Cimetidine, ranitidine, tricyclic antidepressants, fluoxetine and fluvoxamine may decrease metabolism, increase drug levels and produce oversedation.[3] Physicians should consult with the MMTP medical staff before prescribing any of these. Alternatives may be available.

LAAM
(LEVO-ALPHA-ACETYLMETHADOL)

LAAM (ORLAAM®) may have some advantages over methadone. LAAM was originally studied more than two decades ago,[20] but approved by the FDA in 1993. LAAM is metabolized into nor-LAAM and dinor-LAAM, which are both longer-acting and more potent metabolites than the parent drug. Thus, LAAM, has a significantly longer half-life. It can be dosed either every other day, or three times per week. This means fewer clinic visits, which helps patients living long distances from clinic, those with other transportation problems, or those caring for children or other relatives. Take-home doses of LAAM are not permitted, and it is not approved for pregnant women. Women being treated with LAAM must agree to regular pregnancy testing or must use effective contraception.

> *Opioid substitution therapies continue to be controversial, although there is more data for their safety and efficacy than for virtually any other treatment in chemical dependency.*

LAAM doses are usually 30-40% higher than methadone doses. In most programs, patients are initially stabilized and titrated to methadone and then converted to LAAM. In others, LAAM is given initially, but it may take patients longer to stabilize, and longer periods are needed between dose changes. Some patients may not tolerate LAAM and request a switch back to methadone, but most prefer the smoother onset of LAAM, as well as the reduced clinic attendance.

LAAM patients who are hospitalized should be converted to methadone by using approximately 70% of the LAAM dose. Neither LAAM nor methadone should be given to patients until 48 hours after the last LAAM dose. Because of the very long half-life and active metabolites of LAAM, an overdose may occur if other long-acting drugs are given, including available long-acting morphine, oxycodone and fentany.[1] If such drugs are required, they should be used with great caution, and the patient switched from LAAM to methadone.

BUPRENORPHINE

Buprenorphine is a semi-synthetic opioid, which acts as a partial agonist. In higher doses it becomes a mixed agonist-antagonist. Buprenorphine is increasingly being used for detoxification[8] and also as a maintenance drug.[9] Experience with the detoxification of more than 1000 opioid-dependent patients indicates that it is very well tolerated, provides a comfortable withdrawal with fewer post-withdrawal symptoms. Disadvantages include the need for intramuscular administration and high cost. Buprenorphine also precipitates withdrawal in patients with high levels of opioids, particularly long-acting forms such as methadone or Oxycontin®. Advantages include rapid onset, little psychoactive effects in currently used doses (for detoxification, 0.3-0.6 mg intramuscularly every 4-6 hours), and little respiratory depression when used alone.

Buprenorphine is being studied as a sublingual tablet, to which the addition of naloxone may reduce the abuse/diversion potential. The maintenance dose will probably be in the 12-16 mg per day range. Its long half-life may allow three times weekly or every other day dosing. Buprenorphine may be an ideal drug for office-based medical maintenance by specially trained or certified providers.

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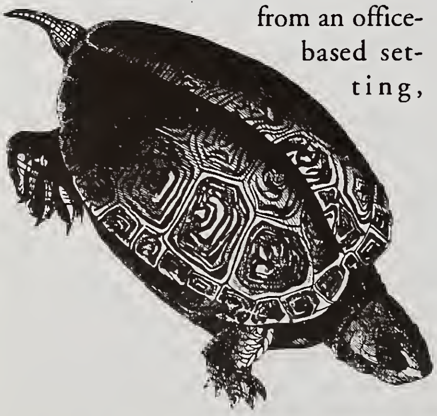
CONCLUSIONS

Opioid-dependent patients are common in all areas of the health care system. While often challenging, they can be successfully treated with outstanding results in social and health functions for both patients and their families. Optimal results require health care professionals to be familiar with the diagnosis and management of opioid dependency. Practitioners must provide referral, ongoing support and care for opioid users' acute and chronic medical, psychological and social issues. This may require examination of our own attitudes and beliefs, to overcome obstacles to competent and compassionate care.

Opioid substitution therapies continue to be controversial, although there is more data for their safety and efficacy than for virtually any other treatment in chemical dependency. Such therapies reduce mortality and morbidity, decrease acquisition and transmission of HIV, hepatitis B and C, improve individual and family function and reduce societal costs. Disparities in quality are being currently addressed by proposed accreditation standards. These standards should assist in providing increased access to, and third party payment for, these programs.

Primary care providers should be able to deal with the challenges of this population. At a minimum, clinicians should be able to (1) provide safe and reasonably comfortable detoxification to opioid dependent patients (2) manage difficult behaviors, and (3) help patients receive long term chemical dependency treatment options. In some cases, the clinician may be able to provide care from an office-based setting, using referral to self-help and family treatment, and following up on the results. Practitioners who develop these skills will contribute greatly to the successes that chemical dependency treatment professionals see daily.



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The Physician's Role in Preventing Alcohol and Drug Abuse

Catherine Dube, EdD

Physicians can play a key role in the prevention of alcohol and other drug abuse and its consequences in the clinical setting. There, the physician can have a powerful effect through screening,[1] diagnosis, counseling,[2,3] and referral.[4] The physician's role in prevention spans all levels of preventive services: 1) primary prevention (interventions aimed at preventing a problem from occurring); 2) secondary prevention (interventions aimed at early identification and treatment); and 3) tertiary prevention (interventions aimed at preventing further complications of a condition or problem).

Physicians regularly practice primary prevention through messages to young patients about avoiding initiation of alcohol or other drug use. Messages should be tailored to the patient's developmental level, social setting and family risk factors. Since age of first use of alcohol and drugs continues to decline,[5] prevention messages need to be provided early in the lifecycle. The American Academy of Pediatrics recommends that prevention counseling begin with parents at the prenatal visit[6] and continue with parents and children throughout childhood. Because the major risk period for initiation of use begins around age 12,[7] anticipatory guidance and other primary prevention messages should intensify in the later elementary school years. Preventive practices should continue throughout the college years when initiation of alcohol and other drug use peaks. (Table 1) Use declines dramatically in the mid to late twenties.[8] An empathic approach, asking about friends and family members' use of alcohol and other drugs, and providing advice on avoidance skills, appears effective.[9] As with other sensitive topics, a non-judgmental approach works best. Focus on the speaker, use open-ended questions and facilitate the patient's discussion of the issue.[10]

Abbreviations Used:

CAGE	4-item alcohol screening scale

Alcohol and other drug problems can develop at any stage of life including among the elderly.[12] Physicians who monitor non-problematic drinking patterns may detect a developing problem. Look for signs that the patient's alcohol consumption exceeds the following levels:

Men:	> 14 drinks per week or > 4 drinks per occasion
Women:	> 7 drinks per week or > 3 drinks per occasion (NIAAA[13])

Table 1.
Primary Prevention Messages for Young Patients and Their Parents [5,9,11]

Stage of Development	Primary Prevention Messages
Prenatal	Ask about parental alcohol, tobacco, illicit drug use, prescription and over-the-counter drug use; Advise parents to avoid or stop use; Refer to treatment if necessary; Discuss teratogenicity, other risks of prenatal exposure, and risks when breastfeeding.
Infancy	Same as above with an emphasis on breastfeeding rather than prenatal exposure; Discuss alcohol/drug effects on parenting skills; Discuss the dangers of second-hand smoke.
Early Childhood	Parents: Discuss alcohol/drug effects on parenting skills and role modeling; Discuss dangers of second-hand smoke; Discuss prevention strategies for parents; Reinforce positive behaviors Child: Assess the child's understanding and feelings about parental or other adult opinions or use; Reinforce attitudes like "Smoking is bad" or "Kids don't drink beer."
Middle & Late Childhood	Parents: Same as above Child: Ask about school functioning; Ask about drug and alcohol prevention programs at school (e.g., DARE, Drug Abuse Resistance Education) Assess understanding and feelings about alcohol and drug use; Teach about the dangers of drug, alcohol and tobacco use; Assess intentions to use alcohol, tobacco or other drugs; Reinforce positive attitudes and avoidance strategies.
Adolescence	Parents: Alert to early warning signs of substance/use abuse. Child: Ask about school functioning; Ask about peer group use; Assess current use; Assess exposure and intentions for future use; Provide factual information about health consequences of alcohol, tobacco and drug use; Encourage avoidance; Reinforce positive attitudes and avoidance strategies.
Late Adolescence	Address adolescent in an adult-to-adult manner; Encourage development of own rules and standards; Warn about the dangers of smoking and drinking too much alcohol/drinking and driving; Supply facts about the dangers of drug use.

Patients with these levels of use need to be advised that they are at risk for developing alcohol problems, and that consumption should be reduced.

For secondary prevention of alcohol and other drug abuse, physicians use screening techniques. The most common screening device for alcohol problems is the 4-item CAGE questionnaire developed by Ewing in 1984.[14] Even with asymptomatic patients, these four brief questions can reliably detect drinking problems that require further assessment. Two “Yeses”” will detect existing alcohol abuse or dependence 74% of the time (sensitivity = 74%). A negative screen (3 or more “Nos”) will rule out alcohol abuse or dependence 91% of the time (specificity = 91%).[15]

C – Have you ever felt you needed to *Cut down* on your drinking?
A – Has anyone ever *Annoyed* you because they criticized your drinking?
G – Have you ever felt *Guilty* about something you've done when drinking?
E – Have you ever needed an *Eye opener* or drink first thing in the morning to get going?

Some authors suggest adapting these questions for other drugs of abuse.[16,17] Substitute “use of (the specific drug in question)” for the word “drinking” in the questions. Specificity and sensitivity of this technique has not been fully established.

The American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders (IV)* contains the diagnostic criteria for substance abuse and dependence. The criteria for Substance Abuse includes a “maladaptive pattern of use” of alcohol and/or other drugs resulting in recurrent, serious clinical impairment or distress within a 12-month period. Distress or impairment must be evidenced by at least one of the following: 1) failure to fulfill role obligations; 2) substance use in hazardous situations; 3) drug or alcohol related legal problems; or 4) continued use despite significant interpersonal or social problems resulting from drug or alcohol use.

> *Because the major risk period for initiation of use begins around age 12, anticipatory guidance and other primary prevention messages should intensify in the later elementary school years.*

* * *

Substance dependence occurs when a maladaptive pattern of use leads to clinically significant impairment or stress including at least three of the following over a 12-month period: 1) recurrent use in spite of related physical or psychological problems; and/or 2) important social and other obligations given up or reduced by use; 3) tolerance to the drug effects; 4) withdrawal symptoms, 5) increased use over time; 6) loss of control over use; or 7) preoccupation with use, obtaining the drug, or time spent recovering from drug effects.[18]

Office-based brief interventions represent an effective tool for tertiary prevention. Understanding the patient's readiness for changing his/her substance use behaviors and/or accepting a referral to treatment is the first step. Prochaska and DiClemente[19] described stages in the patient's decision-making process: 1) precontemplation - the patient does not consider use to be a problem and is not considering change; 2) contemplation - the patient recognizes substance use to be a problem, and is considering change

Table 2.
U.S. Preventive Services Task Force Recommendations: Screening for Problem Drinking[20]

Screening for Problem Drinking

Which Patients	Clinical Approach
All Adult and Adolescent Patients	• Obtain history of alcohol use • Screening questionnaires may be helpful to assess the likelihood of problem or hazardous drinking: — CAGE — AUDIT (Alcohol Use Disorders Identification Test)[21] • Follow up with further assessment as needed All Pregnant Patients All of the above plus: • Questions on tolerance might be helpful • Counseling on harmful effects of drinking on the fetus
All Pregnant Smokers	All of the above plus: • Counseling on the combined effects of smoking and drinking
Patients with Alcohol Dependence	• Refer to treatment
Patients with Alcohol Abuse or Hazardous Drinking	• Brief advice • Feedback about evidence and consequences of drinking • Advice to reduce consumption • Plans for regular follow-up
All Patients who Drink	• Counseling on dangers of drinking and driving or other dangerous activities
Patients Under Legal Age	• Counseling discouraging use of alcohol

sometime in the future; 3) ready for action - the patient wants to take action but has no concrete plans; 4) action - the patient has a concrete plan and begins taking steps to implement the plan; and 5) maintenance - the patient successfully addressed the problem, and maintains new, healthy behaviors; 6) relapse - the patient slips back to a previous stage of change.

To assess a patient's stage of change, begin with an open-ended question: "How would you feel about (quitting, obtaining treatment, etc.)?" The patient's response may provide you with all the information you need. If more precise questions are required, you may ask:

1. Have you ever (quit, obtained treatment, etc.) in the past?
2. Do you think your (drinking, drug use) is a problem for you?
3. Have you been thinking about (quitting, getting treatment, etc.)?
4. Would you consider (quitting, getting treatment, etc.) now?
5. Do you have a plan/can we make a plan today to help you (quit, start treatment, etc.)?
6. Have you started any part of your plan to (quit, start treatment, etc.)?
7. Have you been successful (quitting, completing treatment, etc.) for more than 6 months?

Table 3
U.S. Preventive Services Task Force Recommendations: Screening for Drug Abuse[20]

Screening for Drug Abuse

Which Patients	**Clinical Approach**
Adult and Adolescent Patients	• Include questions about drug use in the history • Be alert to signs and symptoms of drug abuse • Ask about use of illicit and legal drugs of abuse
Older children, Adolescents and Young Adults	• Consider use of inhalants
Patients Who Report Drug Use	Establish a trusting relationship and assess respect confidentiality • Quantity, frequency, patterns of consumption • Adverse consequences of drug use
All Pregnant Patients	Same as adult patients above plus: • Advise about the potential risks to the fetus • Advise about the risks of drug use when breastfeeding
Pregnant Patients with Signs and Symptoms of Potential Drug Use	• Urine testing may be appropriate
Pregnant Patients with a History of Drug Use	• Periodic urine testing may help monitor and encourage abstinence
Pregnant Patients Who Abuse Drugs	• Advise about the importance of regular prenatal care • Refer for treatment
Patients who Report Potentially Harmful Use of Drugs	• Inform of risks of continued use • Advise to cut down or stop drug use • Develop a tailored treatment plan
Patients with Drug Dependence	• Refer to treatment
Patients who Continue to Inject IV Drugs	• Screen periodically for HIV infection • Counsel on techniques to reduce risk of spreading infection through IV drug use (e.g., clean needles and works, never share or reuse needles/works, etc.)

Note: Use of routine urine or blood testing is not recommended as the primary method of detecting drug use in asymptomatic adults including pregnant women.

Users who answer "yes" to question 1 are in "Relapse" and their new stage of readiness needs to be assessed. A negative answer to question 2 indicates "Precontemplation." Other stages would be indicated as follows:

Contemplation: 2 = Yes; 3 = Yes; 4 = No
Ready for Action: 5 = Yes; 6 = No
Action: 6 = Yes; 7 = No
Maintenance: 7 = Yes

The physician, recognizing the patient's stage of readiness, customizes advice. For example, if a patient is in Precontemplation it would be appropriate to discuss consequences of use and link these problems in the patient's life with use of alcohol or other drugs.

These interventions can work in combination with a supportive and empathic approach. Confrontation without empathy and support typically fails.[2] Miller and Rolnick describe the five principles of "motivational interviewing" for helping patients change addictive behavior: 1) express empathy - reflective listening to understand the patient's perspectives and emotions without judging them; 2) develop discrepancy - point out and amplify the differences between the patient's current behavior and ultimate personal goals; 3) avoid argumentation - head-to-head argumentation generates resistance; 4) roll with resistance - reframe the patient's comments toward change, offer alternatives and negotiate mutually acceptable solutions; 5) support self-efficacy - empower the patient to feel that he or she has what it takes to make necessary changes, cope with the situation, overcome obstacles or follow through with the referral or treatment plan.[2]

RECOMMENDED CLINICAL PRACTICES FOR ALCOHOL AND DRUG ABUSE PREVENTION

The U.S. Preventive Services Task Force describes the complete range of evidence-based clinical practices for alcohol and other drug abuse prevention. The Task Force rigorously reviewed current research supporting and refuting each preventive practice related to alcohol and drug abuse screening. Their "science-based" recommendations represent "the culmination of over four years of literature review, debate, and synthesis of critical comments from expert reviewers.[20] (Tables 2 and 3).

CONCLUSION

Physicians play an important role at every level of clinical prevention of alcohol and other drug problems. As physicians employ these preventive practices, they should look beyond alcohol and illicit drug use, to prescription and over-the-counter drugs that may result in abuse or dependence, and to the potentially deadly gasoline, glues, and aerosols that children and adolescents may inhale. Routine histories and screening for all patients will provide the physician with the information to detect problems. Primary prevention messages to all youth, young adults and non-problematic drinkers will bolster the patients' own defenses against alcohol and other drug problems. Use of intervention and counseling techniques will improve physicians' effectiveness when assisting patients in need of preventive or treatment services.

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The Impaired Practitioner: Substance Abuse and Unprofessional Conduct in Rhode Island

Milton Hamolsky, MD

The Rhode Island Board of Medical Licensure and Discipline protects citizens from the unprofessional conduct of physicians. Established by law in 1987, the Board (1) ensures that all practicing physicians have the appropriate education, training and credentials to be licensed, (2) investigates and adjudicates complaints alleging unprofessional conduct by licensed medical and osteopathic physicians, (3) publicly disseminates findings and sanctions related to unprofessional conduct. The Board consists of six physicians and six public members and is chaired by the Rhode Island Director of Health.

The enabling statute (RIGL 5-37-5.1) lists 30 specific categories of actions which constitute "unprofessional conduct." Three areas relate to substance abuse: (1) Dependence upon controlled substances, habitual drunkenness or rendering unprofessional services to a patient while the physician or limited registrant is intoxicated or incapacitated by the use of drugs; (2) Promotion by a physician or limited registrant of the sale of drugs, devices, appliances, or goods or services provided for a patient in such a manner as to exploit the patient for the financial gain of the physician or limited registrant; (3) Violating any state or federal law or regulation relating to controlled substances.

> *The enabling statute (RIGL 5-37-5.1) lists 30 specific categories of actions which constitute "unprofessional conduct."*

PROCESS FOR REVIEWING COMPLAINT ABOUT PHYSICIAN SUBSTANCE ABUSE

Except for the diversionary program option discussed below, the Board treats substance abuse cases the same as other allegations of unprofessional conduct. It conducts a brief inquiry, informs the affected physician and refers the case to a subcommittee for investigation. If the subcommittee finds "no unprofessional conduct" and

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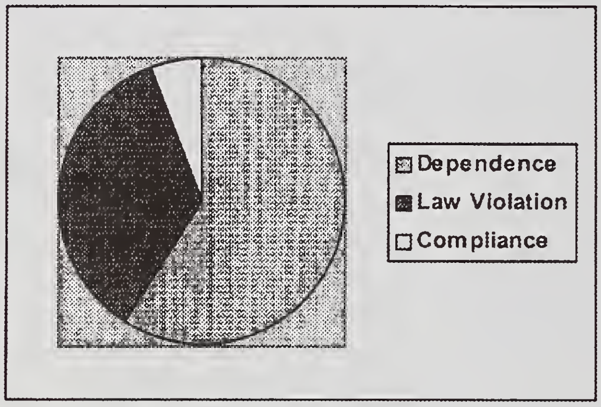
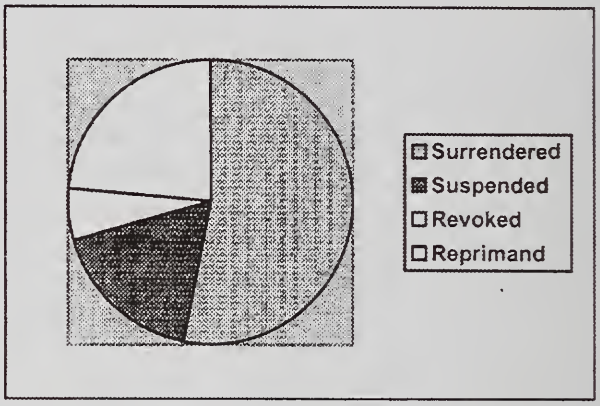
Figure 1
Basis of Unprofessional Conduct Regarding Substance Abuse
(N=17)



Source: Board of Medical Licensure and Discipline 1987-1998

Figure 2
Penalties for Unprofessional Conduct Relating to Substance Abuse
(N=17)



Source: Board of Medical Licensure and Discipline 1987-1998

the remaining Board members concur, the case closes. All proceedings remain confidential. However, if the subcommittee finds "unprofessional conduct," the affected physician has two options. He/she may sign a consent order detailing the facts, legal findings and recommended sanctions - pending approval by the Board. Or the physician may choose a quasi-judicial administrative hearing. The hearing committee renders the decision, issues a written order and imposes sanctions. Decisions may be appealed to the Superior Court.

The Board refers many cases involving physician impairment to the Rhode Island Medical Society's Committee for Physicians' Health. This diversionary program is modeled after those for physicians and attorneys in other states. Each referral is unique and often involves evaluation, recommendations for management, a contractual arrangement, and monitoring by the Board. If the licensee completes the program satisfactorily, the Board closes the pending case. Neither the disciplinary allegation nor the licensee participation in the program becomes public information. To date, only one licensee who entered the program failed to show satisfactory progress.

DATA ON RHODE ISLAND PHYSICIANS

Over the past 11 years the Board reviewed complaints alleging *primary* alcohol and/or drug abuse in 30 of the 3,031 cases opened and investigated by the Board. Estimates from other state/local Boards vary widely. Compared to Rhode Island, some states indicate that a higher percentage of disciplinary cases involve allegations of alcohol or chemical dependency. Physicians in Rhode Island may be more reluctant to report colleagues. Substance abuse contributes to other allegations of professional misconduct such as negligence, abandonment, professional or mental incompetence.

Of the 30 cases investigated by the Rhode Island Board, a few represented cases reported by other states where the licensee also carried a Rhode Island license. In these cases, the Board usually requires the same action(s) and sanction(s) imposed by the other sovereign state if the involved action(s) would have represented unprofessional conduct in Rhode Island.

Between 1987 and 1998, the Board found unprofessional conduct related to a primary allegation of abuse of controlled substances in 17 cases. Ten cases involved "dependence on controlled substances," six represented a violation of State or Federal law (e.g., pre-signing prescriptions for narcotic drugs), and 1 addressed a failure to adhere to the contractual arrangement with the Committee on Physicians' Health. (Figure 1) Out of these 17 findings of unprofessional conduct, 9 culminated in voluntary surrender of the license, 3 led to a suspension, 1 resulted in revocation and 4 produced a reprimand with referral to the Committee on Physiciansí Health. (Figure 2)

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Physician Leadership on National Drug Policy: Advocacy for an Effective Drug Policy

David C. Lewis, MD

The turn of the Twentieth Century was not an easy time for the medical profession. Major reforms occurred in the quality of medical education and licensure standards. Drug policy tested the credibility of medical practitioners. Physicians routinely prescribed commonly-used habit-forming drugs—principally opium and morphine. Patent medicines, often zealously recommended by physicians, proved ineffective and contained both harmful ingredients and habit-forming drugs.

The adverse public reaction to these nostrums and quackery, prodded by muckraking journalists, encouraged medicine to get organized. The American Medical Association (AMA) launched a broad attack against patent medications and exposed physicians who supported their unfounded health claims. The AMA sought to rid the profession of errant physicians and supported tough regulation, such as the 1906 Pure Food and Drugs Act.

The landmark Harrison Narcotic Act of 1914 restricted medical practice by legally prohibiting the medical prescription of narcotics to maintain addicts. In response, an "underworld" of heroin and cocaine addicts soon emerged; addicts were widely disparaged and stigmatized as fiends and criminals. Few physicians tried to treat this group of "underworld" addicts. In an attempt to resolve these issues, the AMA drew a distinction between strictly medical addicts - those with diseases - and others such as correctional cases, mental defectives, and social misfits.[1]

Enforcement of the Harrison Act further separated medical practice from addiction care. Drug prohibition agents zealously prosecuted physicians who continued to treat addicts. By the early 1920s, physicians had largely relinquished their role in the care of the addicted. Criminal justice measures and law enforcement became the key national strategy for dealing with substance abuse.

The inaction of medical leaders in the early 20th century set the stage for today's situation. Law enforcement perspectives dominate federal and state drug policy, despite the fact that drug addiction can be treated through medical and public health approaches. This occurred because physicians, who are most able to bring a medical and public health perspective to the policy arena, abdicated their leadership role. The stigma that followed addiction through the decades goes largely unchallenged; yet few other conditions affect the day-to-day practice of medicine to this magnitude. Now is the time to reassert a policy role for medicine in the treatment of patients and families affected by substance abuse.

Abbreviations Used:

AMA	American Medical Association
FDA	Food and Drug Administration
HHS	Health and Human Services
PLNDP	Physician Leadership on National Drug Policy

Although addicted persons are among the highest users of medical care, only 5% to 10% of these costs come from addiction treatment. Medical problems triggered by the addiction contribute the rest.

The Physician Leadership on National Drug Policy (PLNDP) has come forth to examine existing drug policies and suggest alternatives. Its thirty-seven members include Lonnie Bristow, MD, past president of the AMA; June Osborn, MD, former chair of the National Commission on AIDS; and David Greer, MD, Dean Emeritus of the Brown University School of Medicine. PLNDP draws from the Reagan, Bush, and Clinton Administrations - including a Commissioner of the Food and Drug Administration (FDA), a Secretary of Health and Human Services (HHS), and a Surgeon General - and editors of leading scientific and medical journals.

PLNDP provides medical and public health viewpoints to the debate, in order to fashion a more effective national policy. On July 8, 1997, it convened at the New York Academy of Medicine and reached a consensus on national drug policy.[2] (See PLNDP Consensus Statement box).

STRATEGIC APPROACH

Having physician leaders focus on national drug policy provides a unique opportunity for re-opening discussion about the desired balance between criminal justice and medical/public health approaches. Many audiences with diverse social and political viewpoints welcome the timing and all-medical constituency of the group.

The project calls for both research and coalition-building. PLNDP prepared extensive research review papers to support the group's policy consensus. The first of these reviews, *Addiction and Addiction Treatment*, analyzed drug treatment outcome research, including the cost-effectiveness of treatment programs. The analysis reviewed over 600 peer research articles and undertook original data analyses. The second review, *Health, Addiction Care, and the Criminal Justice System*, looked at the impact of addictions on courts, prisons and related programs.

The summaries follow:

ADDICTION AND ADDICTION TREATMENT

(MARCH 17, 1998)

This document represented an integrated effort to provide a scientific foundation for drug policy alternatives. It discussed the similarities of drug addiction to other chronic illness. It challenged physicians and other health professionals to review their role in addressing drug addiction and its consequences.[3]

Prevalence and costs of addictions relative to other chronic diseases: Using prevalence and economic data, the report compared drug addiction to other common chronic medical conditions. It showed that the combined prevalence of alcohol and drug dependence is about as great as that of heart disease, but addiction accounts for more lost productivity than heart disease and diabetes combined. The total annual cost per affected person for drug addiction is greater than stroke, diabetes, or heart disease. However, improvements in employment status and work productivity far outweigh the costs of drug treatment. Figure 1 shows the cost of addiction treatment compared with incarceration.

> *For more than a decade, studies have demonstrated that addiction treatment reduces overall medical care utilization and costs.*

Cost-effectiveness of treatment: This report presented data on how to enhance treatment effectiveness at a reasonable cost. In addition to considerable savings in short and long term medical costs, addiction treatment provides other benefits. For both the individual and society, interpersonal conflicts, injuries, and crime decrease, while work productivity increases.

Returns on the drug addiction treatment investment: Alcohol and drug addiction contributes to the incidence and severity of many medical conditions. Although addicted persons are among the highest users of medical care, only 5% to 10% of these costs come from addiction treatment. Medical problems triggered by the addiction contribute the rest. For more than a decade, studies have demonstrated that addiction treatment reduces overall medical care utilization and costs.

Drug dependence as a treatable medical illness: This report compared addiction treatment to treatment of other chronic medical conditions such as hypertension, diabetes, and asthma. Drug dependence meets the criteria for a treatable, chronic, consistently diagnosable medical condition. Treatment outcomes compare favorably to other chronic conditions. Comparisons of

PLNDP

CONSENSUS STATEMENT

Addiction to illegal drugs is a major national problem that creates impaired health, harmful behaviors, and major economic and social burdens. Addiction to illegal drugs is a chronic illness. Addiction treatment requires continuity of care, including acute and follow-up care strategies, management of any relapses, and satisfactory outcome measurements.

We are impressed by the growing body of evidence that enhanced medical and public health approaches are the most effective method of reducing harmful use of illegal drugs. These approaches offer great opportunities to decrease the burden on individuals and communities, particularly when they are integrated into multidisciplinary and collaborative approaches. The current emphasis – on use of the criminal justice system and interdiction to reduce illegal drug use and the harmful effects of illegal drugs – is not adequate to address these problems.

The abuse of tobacco and alcohol is also a critically important national problem. We strongly support efforts to reduce tobacco use, including changes in the regulatory environment and tax policy. Abuse of alcohol causes a substantial burden of disease and antisocial behavior which requires vigorous, widely accessible treatment and prevention programs. Despite the gravity of problems caused by tobacco and alcohol, we are focusing our attention on illicit drugs because of the need for fundamental shift in policy.

AS PHYSICIANS, WE BELIEVE THAT:

It is time for a new emphasis in our national drug policy by substantially refocusing our investment in the prevention and treatment of harmful drug use. This requires reallocating resources toward drug treatment and prevention, utilizing criminal justice procedures which are shown to be effective in reducing supply and demand, and reducing the disabling regulation of addiction treatment programs.

Concerted efforts to eliminate the stigma associated with the diagnosis and treatment of drug problems are essential. Substance abuse should be accorded parity with other chronic, relapsing conditions insofar as access to care, treatment benefits, and clinical outcomes are concerned.

Physicians and all other health professionals have a major responsibility to train themselves and their students to be clinically competent in this area.

Community-based health partnerships are essential to solve these problems.

New research opportunities produced by advances in the understanding of the biological and behavioral aspects of drugs and addiction, as well as research on the outcomes of prevention and treatment programs, should be exploited by expanding investments in research and training.

Over the next year, *Physician Leadership on National Drug Policy* will review the evidence to identify and recommend medical and public health approaches that are likely to be more cost-effective, in both human and economic terms. We shall also encourage our respective professional organizations to endorse and implement these policies.

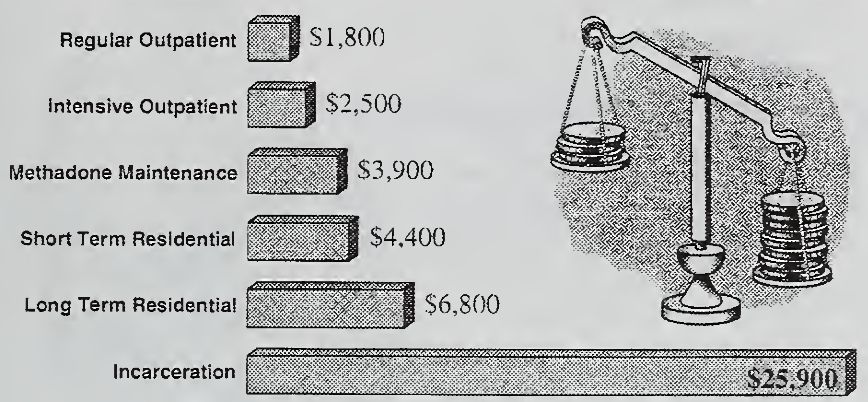

Figure 1. Weighing the Costs: Annual Cost Per Drug Addict (from the PLNDP National Office at Brown University, Prepared by Norman Hoffmann, Ph.D.)

medication and behavioral compliance revealed that addicted patients comply at a rate similar to that of patients receiving treatment for diabetes, asthma, and hypertension.

Health, Addiction Care, and the Criminal Justice System (November 10, 1998)

This report found that the best new programs reduce drug use, crime, and re-arrest rates. Close collaboration between the criminal justice system, community public health agencies, cognitive and behavioral counselors, drug treatment specialists, health care providers, and employment specialists improves success dramatically.

Drug Courts and Long-term Residential Treatment: Drug courts exist in over 275 US jurisdictions. According to this study, drug court programs lower recidivism rates, decrease costs of addiction and drug use, and improve gainful employment of the participants. Improvements occur both during their participation and after graduation. Successful drug courts work well with first-time and chronic offenders. The most important components of success include close, efficient, team-based collaboration between the drug court and the community's treatment systems.

Prison Drug Treatment: The prisoner's self-selection contributes to retention in and compliance with the treatment regimen. An ideal treatment time is three to nine months, but one expects several episodes of treatment, aftercare, and relapse because of the nature of drug dependency. Therapeutic communities and cognitive behavioral treatments stood out as successful treatments for hard-core prison inmates displaying predatory behavior.

Parolees and Community After-care: Two-thirds of probationers reported serious drug and alcohol problems. Only 17% of these probationers have access to substance abuse treatment once they leave prison. Long-term resi-

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dential (LTR) treatment can make major improvements. In the best LTR programs, weekly cocaine use dropped by 67%, weekly heroin use by 65%, and heavy drinking dropped by 53%. Only about 27% of "aftercare graduates" returned to custody, compared to 75-84% from comparison group.

Juvenile Substance Abuse Treatment: The best programs cut recidivism by 30% or more. Conduct and delinquency problems have a pervasive, long-standing association with youth drug involvement. While the relationship is complex, most studies indicate that the conduct problems precede the development of substance abuse problems. Delinquency is a key risk factor for adolescent substance use and abuse, rather than an outcome of drug use. Cessation of delinquency behaviors during adolescence and young adulthood is associated with a discontinuation of substance abuse.

PHYSICIAN ASSOCIATES OF THE PLNDP

PLNDP established an Associates group to relate these research reports with the personal experiences and practices of physicians across the country. Staff corresponded with practicing physicians, telling them of the project, asking for their opinion of the PLNDP consensus statement, and offering the chance to become an Associate. A Physician Associate agrees with the consensus statement of the Physician Leadership on National Drug Policy and receives periodic materials about the progress and activities of the PLNDP. Over 95% of the physician-respondents supported the consensus statement. Several thousand physicians joined as Associates. PLNDP also established a website and newsletter.

VIDEOTAPED REPORTS OF THE RESEARCH SUMMARIES AND ACTION INITIATIVES

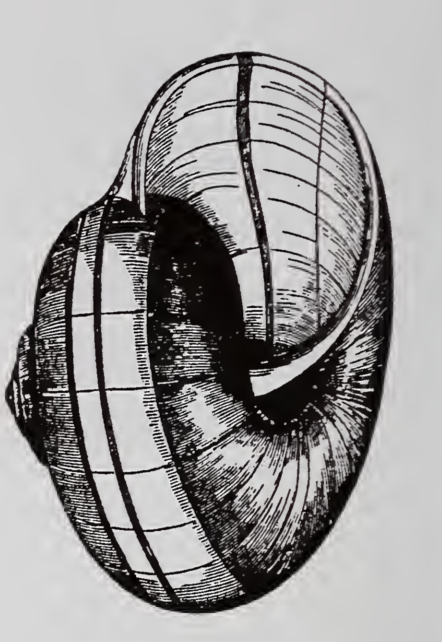
PLNDP provides the educational tools to build consensus and networking among members and associates. First, PLNDP produced videotapes of each research presentation. (The videotape, "Drug Addiction: The Promise of Treatment, " is available from the PLNDP National Office.) Second, PLNDP distributed an action plan to all members and associates. This plan suggests specific actions for implementing the consensus goals. One action advocates medical parity: "Move towards parity of health care access and insurance benefits for addiction treatment: Parity refers to equal treatment for substance abuse when compared to other chronic conditions. This includes insurance coverage for treatment as a benefit in health care and providing equal access to treatment." Other topics include obtaining endorsements of the PLNDP consensus statement, learning about substance abuse treatment and the criminal justice system, incorporating substance abuse training in medical education, and supporting relevant community coalitions.

SUMMARY

The founders of the PLNDP envisioned an active and committed group willing to tackle the potentially controversial area of national drug policy. The group already initiated a serious national debate about drug policy, inside and outside of the medical profession. Several national professional societies moved beyond their existing policies to embrace components of the PLNDP initiative.

The group meets again in the summer of 1999 to share goals and articulate the next steps in research and advocacy. This meeting invites leaders in law, the enforcement community, legislators, community coalition leaders, and experts in addiction medicine and addiction psychiatry. PLNDP leaders expect that the subsequent coalitions will affect the course of discussion about the nation's drug policy far into the future.

- The PLNDP WEB SITE: <www.caas.brown.edu/plndp>
- To request the 18-minute video, "Drug Addiction: The Promise of Treatment:" phone (401) 444-1817.
- Membership information: PLNDP National Office phone: (401)444-1817, e-mail: plndp@brown.edu



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Colorectal Cancer Screening and Prevention

Raymond Maxim, MD

Colorectal cancer is the third leading cause of cancer death for both men and women in this country. The estimated lifetime risk of colorectal cancer death is 2.6%.[1] It accounts for almost 56,000 deaths each year. The Centers for Disease Control and Prevention (CDC) project that there will be over 130,000 newly diagnosed cases in 1998. However, if current trends continue only 35% of these cancers will be discovered in a localized stage where the five year survival is over 90%. The rest will be found after progression to regional disease (38%) or already metastatic disease (22%) where the survival rates (69% for regional and 9% for metastatic disease) are much less encouraging.[2]

The natural history of colon cancer is a slow progression from adenomatous polyp to cancer over a period of many years. This makes early detection and treatment possible, but only if adequate screening takes place. Unfortunately, despite the demonstrated effectiveness of fecal occult blood testing (FOBT) and flexible sigmoidoscopy, the 1992 National Health Information Survey reported only 26% of individuals over the age of 50 reported ever having FOBT in the past three years. Only 33% of those in the studied age range had ever been screened with flexible sigmoidoscopy.

Recommendations for screening strategies are not as clear cut as the demonstrated effectiveness of screening. There are multiple guidelines in the recent literature including those from The American Gastroenterological Association[3], the United States Preventive Task Force[4], and The American Cancer Society. They all agree on some basic principles. First, a screening strategy not pursued results in no gain. All persons over the age of 49 should be screened. Those individuals at high risk should be screened earlier and more aggressively than those with normal risk. FOBT should probably be part of any strategy despite its low sensitivity and specificity because of its comparatively low cost.

A change to the Medicare benefits effective January 1, 1998, covers screening for colorectal cancer. Fecal occult blood tests will be covered for beneficiaries older than age 49 once every 12 months. This is in addition to other screening methods. Flexible sigmoidoscopy will be covered once every 48 months for those at normal risk for colorectal cancer. Screening colonoscopy will be covered once every 24 months for those at high risk for colorectal cancer. The Health Care Financing Administration (HCFA) defines high risk as:

Abbreviations Used:

CDC	Centers for Disease Control and Prevention
FOBT	fecal occult blood testing
HCFA	Health Care Financing Administration

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COLORECTAL SCREENING GUIDELINES AVAILABLE ON THE INTERNET:

U.S. Preventive Task Force
http://158.72.20.10/pubs/guidecps/default.htm

American Cancer Society
http://www.cancer.org

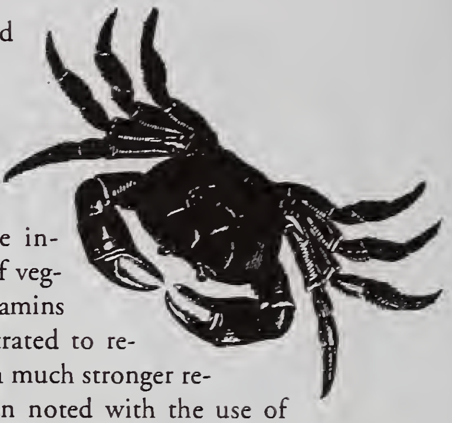
Interdisciplinary Task Force
http://www.gastro.org/colcancer

Health Care Financing Administration
http://www.hcfa.gov

- a first degree relative with colorectal cancer or an adenomatous polyp;
- a family history of familial adenomatous polyposis;
- a family history of hereditary nonpolyposis colorectal cancer;
- a personal history of adenomatous polyps; or
- personal history of colorectal cancer; or
- inflammatory bowel disease

A screening barium enema for those not at high risk will be covered every 48 months as an alternative to flexible sigmoidoscopy and every 24 months for those at high risk as an alternative to colonoscopy.

Just as the prolonged natural history of colorectal cancer provides ample opportunity for screening it also makes primary prevention feasible. Like heart disease, there are both modifiable and unmodifiable risk factors. For the unmodifiable risk factors (inflammatory bowel disease, certain genetic syndromes, family history, or personal history of colon cancer) the strategy is screening. For the modifiable risk factors, lifestyle changes and minor interventions could prevent an estimated 50% of cancers. Persons with a sedentary lifestyle are at greater risk and the recommendations for increased physical activity are consistent with the current cardiovascular risk factor reduction recommendations. Obesity also shares the association of increased risk that is independent of sedentary lifestyle. The Nurses' Health Study identified the amount of dietary red meat consumption as another strong association with the development of colon cancer. Daily consumption of red meat made the likelihood of cancer 2.5 times greater than those who only ate red meat once a month.[5]



Dietary folate intake in the form of vegetables or multivitamins has been demonstrated to reduce risk. In fact, a much stronger relationship has been noted with the use of multivitamins. A diet that is high in vegetables has a protective effect independent of the folate content.

It was previously thought that a diet high in fiber was protective, although recent evidence suggests that it may not reduce the risk for colon cancer.[6] We still can recommend a high fiber diet for its cardiovascular benefits. A reduction in alcohol intake and smoking cessation are also helpful in reducing colorectal cancer risk. Aspirin in individuals with no contraindications exerts a protective effect when used long term. Supplemental calcium has some evidence base to suggest a beneficial effect in recent literature.[7]

In summary, colorectal cancer is one of those disease processes where there exists considerable knowledge base. We have effective screening tools, effective interventions in early disease and we know how to prevent a large proportion of the disease burden. All we have to do now is put into practice what we already know.

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Health by Numbers

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Jay S. Buechner, PhD, and William J. Waters, Jr., PhD

Alcohol and Drug Use Among Rhode Island High School Students, 1997

Jana E. Hesser, PhD

The use of alcohol and other addictive substances by adolescents presents a major national health concern.[1] Illicit drug use among high schools students and other young adults in America exceeds that of any other industrialized nation in the world.[2] The prevalence of drug use among adolescents in the United States increased throughout the 90s. A recent national study indicates that the upward trend reversed direction in 1998.[3] This paper presents data on the self-reported use of alcohol and other addictive substances among Rhode Island high school students in 1997.[4]

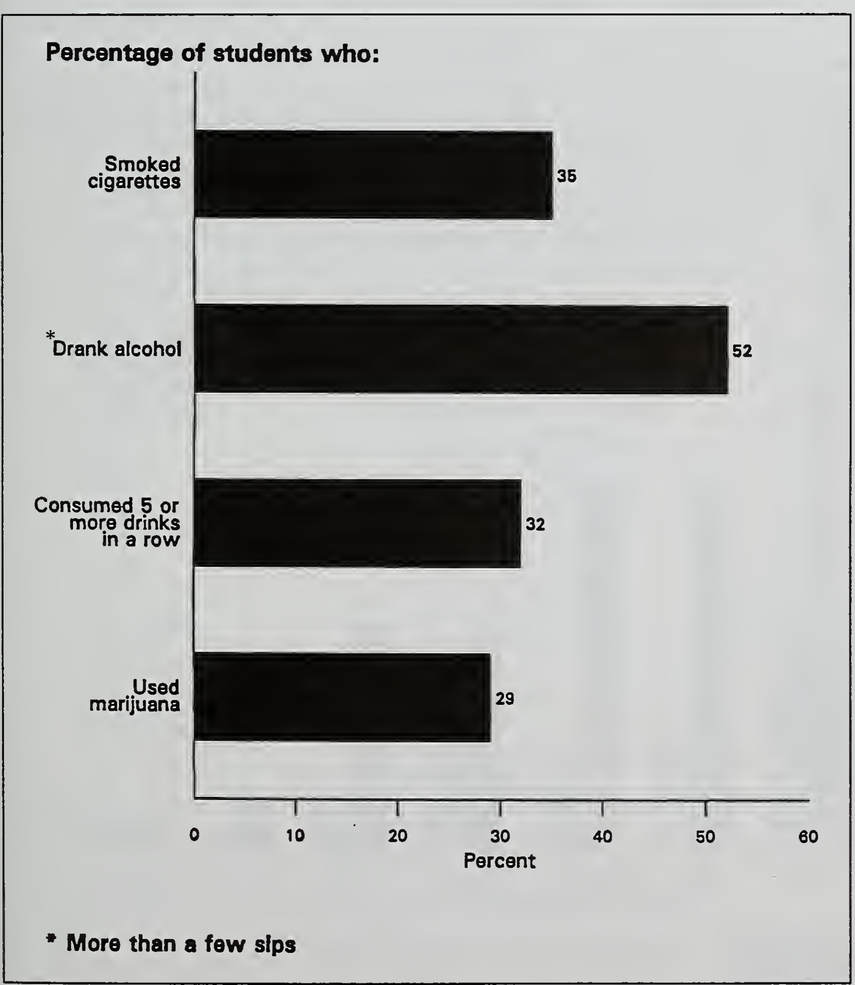


Figure 1. Reported use of cigarettes, alcohol, and marijuana during the 30 days preceding the survey, by students in grades 9 – 12, Rhode Island, 1997.

Abbreviations Used:

CDC	Centers for Disease Control and Prevention
YRBS	Youth Risk Behavior Survey

Methods

The Centers for Disease Control and Prevention (CDC) sponsors the Youth Risk Behavior Survey (YRBS) in Rhode Island and other states to monitor priority health risk behaviors. States conduct this voluntary, anonymous and confidential survey every other year among a randomly selected sample of students in grades 9 - 12. Students answer questions about a variety of health risk behaviors.

Rhode Island conducted the YRBS in May and June of 1997 among a statewide random sample of 1,528 students in grades 9 through 12. Survey administration procedures, established by the CDC, protect student privacy and confidentiality. A number of questions inquired about the use of cigarettes, alcohol, marijuana, cocaine, inhalants, other illegal substances, and needle use to inject illegal drugs into the body, during the students' lifetime and/or during the 30 days preceding the survey.

Results

The YRBS reveals a substantial use of cigarettes, alcohol, and other drugs among Rhode Island high school students. These rates are similar to national rates.[2] Rhode Island students use alcohol more than any other substance. More than three of every four students (78%) have consumed at least one drink of alcohol (other than a few

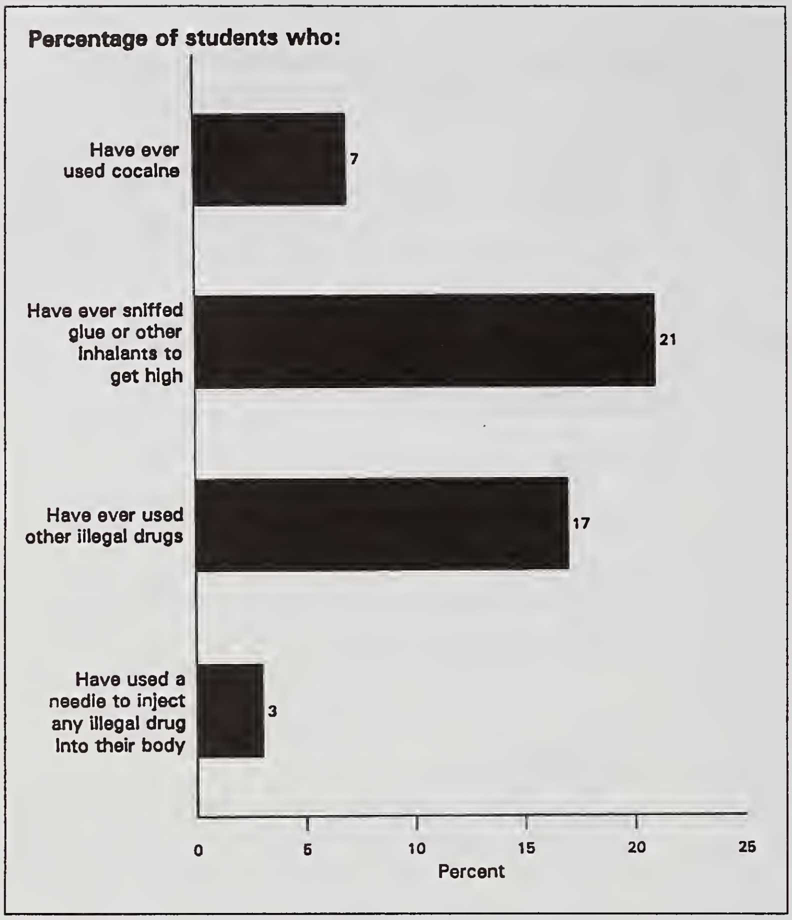


Figure 2. Reported lifetime use of other illegal substances by students in grades 9 – 12, Rhode Island, 1997.

sips) in their life. One of every two students (52%) had one or more drinks during the 30 days preceding the survey. About a third (32%) consumed five or more drinks in a row (binge drinking) once or more during the prior month (Figure 1). About one of every three students (34%) took their first drink before age thirteen. Males demonstrate greater risk for initiating alcohol use before age 13 and for binge drinking than females.

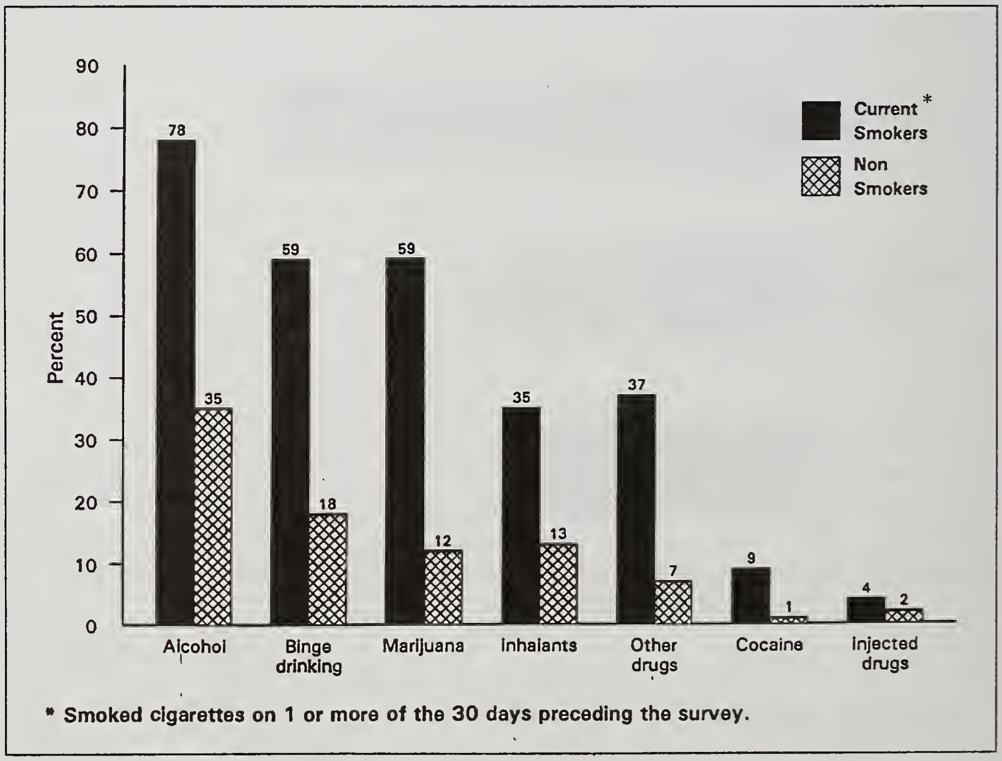
Cigarettes take second place among commonly used substances. A little more than a third (35%) of the students smoked cigarettes on one or more of the 30 days preceding the survey (Figure 1). Two of every three students (69%) have tried cigarette smoking (even one or two puffs); 24% smoked a whole cigarette for the first time before age 13.

Marijuana is used almost as commonly as cigarettes. Nearly a third (29%) used marijuana once or more in the month preceding the survey (Figure 1). Almost half of the students (47%) have used marijuana one time or more in their life and 11% first tried marijuana before age 13.

Cigarette, alcohol, and marijuana use increases with grade level. Twelfth-graders reported the highest use of each substance. In the month prior to the survey, nearly half (49%) of 12th graders smoked cigarettes; 2 of every 3 (67%) had drunk alcohol; and 40% had used marijuana at least once.

While students use alcohol, cigarettes, and marijuana most commonly, a substantial percentage also reported lifetime use of other addictive substances (Figure 2): 21% have sniffed glue or other inhalants to get high; 17% have used other illegal drugs (such as LSD, PCP, ecstasy, mushrooms, speed, ice, or heroin); 7% have used cocaine; and 3% have used a needle to inject an illegal drug into their body.

The use of alcohol and other drugs often begins early in life. Cigarettes are often the first addictive substance adolescents try. Students who smoke cigarettes display a greater risk of using other addictive substances than non-smokers (Figure 3). Use of cigarettes may be easier to detect than the use of other substances. If a young person smokes cigarettes, the probability of present or future alcohol, marijuana, or other drug use increases. If a physician knows that an adolescent patient smokes, the physician should be alert to other possible drug use.



* Smoked cigarettes on 1 or more of the 30 days preceding the survey.

Figure 3. Reported drug use by students who are current smokers or non-smokers, grades 9 – 12, Rhode Island, 1997.

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Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

New Guidelines for the Prevention and Treatment of Osteoporosis

John P. Fulton, PhD

In early November, 1998, the National Osteoporosis Foundation (NOF) released its *Physician's Guide to Prevention and Treatment of Osteoporosis.*[1] The *Guide* contains recommendations developed in collaboration with the following organizations:

- American Academy of Orthopaedic Surgeons
- American Academy of Physical Medicine and Rehabilitation
- American Association of Clinical Endocrinologists
- American College of Obstetricians and Gynecologists
- American College of Radiology
- American College of Rheumatology
- American Geriatrics Society
- American Society for Bone and Mineral Research
- Endocrine Society

Conrad Johnston, Jr., MD, of Indiana University, NOF's Vice President, chaired the development committee which produced the 30-page *Guide* and a companion *Pocket Guide to Prevention and Treatment of Osteoporosis,* which summarizes the recommendations in a handy format.

In a November 5 news release which accompanied issuance of the Guide,[2] the NOF described the following key recommendations:

"The recommendations specify that the following people should have a BMD [bone mineral density] test: women who are 65 and older, regardless of other risk factors; postmenopausal women who have one or more risk factors for osteoporotic fracture (besides menopause); and all postmenopausal women who have a fracture. All postmenpausal women who present with vertebral or hip fractures, two of the most common osteoporosis fracture sites, should be considered candidates for osteoporosis treatment."

WHAT'S NEW?

The *Physician's Guide to Prevention and Treatment of Osteoporosis* improves upon the now out-of-date *Osteoporosis: A Guide to Diagnosis, Prevention, and Treatment,* published by the NOF in 1992.[3] The new volume benefits from the collaboration of many disciplines and the accumulation of medical experience in the 1990s, including the use of alendronate as an effective treatment option.

Abbreviations Used:

BMD	bone mineral density
EFO	European Foundation for Osteoporosis
NOF	National Osteoporosis Foundation

The 1998 *Guide* gives clear guidance for the use of the BMD test. Its recommendations go further than those published by the U.S. Clinical Services Task Force in 1996.[4] At that time the Task Force wrote: "There is insufficient evidence to recommend for or against routine screening for osteoporosis with bone densitometry in postmenopausal women. Recommendations against routine screening may be made on other grounds. ... For those high-risk women who would consider estrogen prophylaxis only to prevent osteoporosis, screening may be appropriate to assist treatment decisions." The *Guide*'s recommendations for the BMD test are broadly consistent with those published in late 1997 by the European Foundation for Osteoporosis (EFO) and Bone Disease.[5] Two key principles underlie the recommendations of both: 1) BMD testing should be based on risk; and 2) BMD testing should be performed only when the results will influence treatment decisions.

Recommendations for intake of calcium and vitamin D have been greatly simplified. *All* persons are advised to consume at least 1200 mg of calcium and 400-800 IU of vitamin D daily.

Recommendations for initiation of treatment are more aggressive than the EFO's guidance on the decision to treat. The 1998 *Guide* suggests initiating therapy for all women with BMD T-scores below –2 in the absence of risk factors,"[1] while the EFO suggests considering treatment in this range "in more elderly with history of fragility fractures."[5]

The 1998 *Guide* clearly prioritizes options for pharma-

HIGHLIGHTS

**** Clear guidance for the use of the BMD test:**

"BMD testing should be performed on:

1. All postmenopausal women under age 65 who have one or more additional risk factors for osteoporosis (besides menopause)
2. All women aged 65 and older regardless of additional risk factors
3. Postmenopausal women who present with fractures (to confirm diagnosis and determine disease severity)
4. Women who are considering therapy for osteoporosis, if BMD testing would facilitate the decision
5. Women who have been on hormone replacement therapy for prolonged periods."

**** Simplified recommendations for intake of calcium and vitamin D:**

For *all* patients

Calcium:	at least 1200 mg/day, including supplements if necessary
Vitamin D:	400-800 IU/day for those at risk of deficiency, including the elderly and those who are chronically ill, housebound, or institutionalized

**** New recommendations for the initiation of treatment:**

"Suggest initiating therapy to reduce fracture risk in women with BMD T-scores below -2 in the absence of risk factors and in women with T-scores below -1.5 if other risk factors are present" such as personal history of fracture as an adult, history of fracture in a first-degree relative, current cigarette smoking, and low body weight (<127 lbs). These cutpoints were defined "in part by analysis of treatment cost-effectiveness."

**** Prioritized options for pharmacologic intervention:**

1. Hormone replacement therapy: "provides the greatest benefit relative to its cost"
2. Alendronate: "a powerful option for those women who meet BMD criteria for treatment but who are unwilling or unable to take HRT"
3. Calcitonin: "a safe but somewhat less effective intervention for osteoporosis" than hormone replacement therapy or alendronate
4. Raloxifene: "approved by the FDA for prevention of osteoporosis, is another alternative for postmenopausal patients."

cologic intervention. Alendronate, introduced for widespread use after the publication of the NOF's 1992 *Guide to Diagnosis, Prevention, and Treatment,* is ranked between hormone replacement therapy and calcitonin for effectiveness in the treatment of osteoporosis, and raloxifene is clearly described as an alternative approach limited to the prevention of osteoporosis in postmenopausal patients.

OBTAINING THE GUIDE

Single copies of the Guide are free, by faxing the following information to (202)223-2237: name, organization, address, city, state, zip code, country, telephone number, fax number. Please request the newly published *Physician's Guide and Pocket Guide.* Orders for multiple copies incur charges. It is suggested that you request information on pricing by fax.

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Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health
Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report
Provisional Occurrence Data from the Division of Vital Records

Vital Events

	Reporting Period		
	September 1998	**12 Months Ending with September 1998**	
	Number	**Number**	**Rates**
Live Births	1,034	13,206	13.3*
Deaths	719	9,862	10.0*
Infant Deaths	(9)	(105)	8.0#
Neonatal deaths	(9)	(83)	6.3#
Marriages	970	7,537	7.6*
Divorces	314	3,233	3.3*
Induced Terminations	373	4,761	360.5#
Spontaneous Fetal Deaths	88	946	71.6#
Under 20 weeks gestation	(82)	(868)	65.7#
20+ weeks gestation	(6)	(78)	5.9#

* Rates per 1,000 estimated population
# Rates per 1,000 live births

Underlying Cause of Death

	Reporting Period			
	March 1998	**12 Months Ending with March 1998**		
	Number (a)	**Number (a)**	**Rates (b)**	**YPLL (c)**
Diseases of the Heart	266	3,251	328.3	3,835.5
Malignant Neoplasms	225	2,493	251.8	6,997.0
Cerebrovascular Diseases	53	683	69.0	734.5
Injuries (Accident/Suicide/Homicide)	31	336	33.9	6,491.0**
COPD	46	476	48.1	347.5***

**Excludes three deaths of unknown age ***Excludes one death of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.
(b) Rates per 100,000 estimated population of 990,225
(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

Necrology, 1998

IRVING A. BECK, MD

Dr. Irving Beck died on January 25, 1998.

A lifelong resident of Providence, Dr. Beck received his bachelor's degree from Brown University and his medical degree from Harvard Medical School, with the highest academic honors. He completed residency training in 1939.

He was an Army veteran of World War II and served as a captain in the Medical Corps. Returning to the United States as a major, he was briefly the deputy chief of medical services at Camp Edwards in Masschusetts.

In 1962 Dr. Beck became chief of the medical service at Miriam and Lying-In (now Women & Infants) Hospitals, and consultant in internal medicine to Rhode Island and Roger Williams Hospitals. In 1962 he was chosen as the first clinical faculty member to counsel students in Brown's new master's degree program in biomedicine. He was president of the Providence Medical Association, governor (for Rhode Island) of the American College of Physicians, president of the Rhode Island Diabetes Association, and president of the American Osler Society.

Dr. Beck was awarded Brown Medical School's W.W. Keen Award "for distinguished achievements and service to the community and to Brown University." In 1986 he was also awarded the Ralph O. Claypode Memorial Award by the American College of Physicians.

He was the husband of Edith (Woodhead) Beck. Besides his wife, he leaves three daughters, two brothers and six grandchildren. He was the father of the late Bernard Beck and Beatrice Beck.

ARTHUR BARNABUS CUDDY, MD

Dr. Arthur Barnabus "Doc" Cuddy died on February 11, 1998.

A lifelong Cranston resident, Dr. Cuddy was an athlete and track star before an automobile accident paralyzed his right leg. After recovering from his injuries, he entered Tufts Medical School and graduated in 1941.

Dr. Cuddy served his internship at Memorial Hospital, and his surgical residency at the Lying-In Hospital, Providence.

During his years of practice, Dr. Cuddy delivered more than 5,000 babies. He was a member of the American Medical Association and the Rhode Island Medical Society.

Dr. Cuddy was an accomplished musician, playing the violin, clarinet and saxophone. He loved sailing and became one of the best-known Herreshoff "S" Class sailors. During the 1950s and 1960s, he and his crew won many Narragansett Bay championships. One of the oldest members of the Rhode Island Yacht Club, he was awarded a lifetime membership.

Dr. Cuddy was the husband of the late Alice (Etherington) Cuddy. He leaves three sons, Arthur B. "Mac" Cuddy and Louis J. Cuddy, both of Warwick, and Michael Cuddy of Cranston; two daughters, Nancy Wood and Patricia Cuddy, both of Warwick; a brother, George Cuddy in Rhode Island; and five grandchildren. He was the brother of the late Helene Dowdall, Mary Bishop and Louis, Jack and Dr. Fred Cuddy.

EDWARD CARDILLO, MD

Dr. Edward Cardillo died on February 14, 1998.

A lifelong resident of Providence, Dr. Cardillo maintained a private practice on Broadway in Providence, and served on the staffs of Women & Infants Hospital, Rhode Island Hospital and Roger Williams Medical Center.

Dr. Cardillo was a graduate of the University of Rhode Island ('42) and received his medical degree from Tufts Medical School ('45). Dr. Cardillo was an Army Air Forces veteran before beginning his practice.

He was a member of the Providence Medical Association and the Rhode Island Medical Society, as well as a diplomat of the American Board of Obstetrics and Gynecology. At Roger Williams Hospital, he had been the director of the division of gynecology. He was chairman of the board of directors of Women & Infants Indemnity Ltd., an insurance company he helped establish, and director of the Dr. Edward Cardillo Health Services Library at Women & Infants Hospital. He served the teaching programs at Tufts and Brown University Medical Schools, and was a trustee of Women & Infants Hospital and Care New England.

Dr. Cardillo was the husband of the late Anna L. (DiZoglio) Cardillo. He leaves three daughters, Donna A. Pensa of Jamestown, Christie A. Velesig of Mashpee, Mass., and Barbara A. Cardillo, PhD, of Los Gatos, Calif.; a sister, Adeline Frantantaro of North Providence; five grandchildren; and a great-grandson. He was the father of the late Cynthia A. Cardillo, and brother of the late Joseph Cardillo.

RICHARD G. MIGNACCA, MD

Dr. Richard G. Mignacca died Monday, February 16, 1998.

Born in Providence, Dr. Mignacca was a Navy squadron medical officer from 1965 to 1967 He was a 1960 graduate of Providence College and a 1964 graduate of Tufts University School of Medicine.

Dr. Mignacca was a diplomat of the American Board of Internal Medicine and the National Board of Medical Examiners. He was chief medical examiner and physician in charge of quality assurance for the determination unit for the State of Rhode Island Department of Disability. Dr. Mignacca served on the staffs at Rhode Island Hospital, St.

Joseph Hospital and Our Lady of Fatima Hospital. He was a clinical assistant professor of medicine and a clinical instructor of medicine at Brown University.

Besides his wife and mother, he leaves three daughters, Donna Salusti of Lexington, Mass., Lisa Mullin of West Hartford, Conn., and Lynn Farren of Seekonk; two sons, Richard S. Mignacca of Somerset and Steven Mignacca of East Providence; and six grandchildren.

JOSEPH L.C. RUISI, MD

Dr. Joseph L.C. Ruisi died on May 3, 1998.

Born in Westerly, Dr. Ruisi received a bachelor's degree from the University of Rhode Island ('37) and a medical degree from Tufts University Medical School ('41).

Dr. Ruisi served as Provincial Medical Officer in the Allied Military forces in Italy during World War II and was discharged in 1945 with the rank of major. He received many decorations for his services, including the Bronze Star.

Dr. Ruisi was a former president of the Westerly Red Cross, and Westerly Cancer Society. He was also a member of the Rhode Island Medical Society, and a member and past president of the Washington County Medical Society.

In addition to his wife, he is survived by two sons, two daughters and seven grandchildren.

GUSTAVO A. MOTTA, MD

Dr. "Gus" Motta died on July 23, 1998.

Born in Providence, a son of the late Adolph J. and Maria Caterena (Alterio) Motta. Dr. Motta was a 1935 graduate of Providence College and a member of its alumni association. Dr. Motta was a graduate of Georgetown University, cum laude, and Georgetown University Medical School in 1940.

Dr. Motta was an Army veteran of World War II, serving in the Medical Corps, Medical Detachment, 101st Field Artillery Battalion. As battalion surgeon, he worked long hours, often under enemy fire.

Dr. Motta maintained a medical office in Providence for 50 years, retiring in 1983. He was inducted as a fellow of the American Board of Family Practice in New York in recognition of his post graduate medical education. He was former president of the Rhode Island Chapter of the American Academy of General Practice and president of the Providence Medical Association. He was a former staff president at St. Joseph Hospital and Our Lady of Fatima Hospital, and received an award for 25 years of service in September of 1981.

Dr. Motta leaves a daughter, two sisters, a brother and several nieces and nephews. He was the father of the late Gustavo A. Motta, Jr.

PHILIP J. LAPPIN, MD

Dr. Philip J. Lappin died on October 10, 1998.

Born in Barrhead, Scotland, the youngest of 10 children, he was a resident of Pawtucket for 40 years, previously living in Central Falls. Dr. Lappin was a graduate of Central Falls High School and Brown University, and graduated from Tufts University Medical School with honors. He opened his family practice office on Broad Street, Central Falls in 1952, retiring in 1995.

Dr. Lappin was on the staff of Memorial Hospital and a past president of its medical staff. He was the founder and director of the Pawtucket Memorial Hospital Home Care Program and a member and former president of the medical staff of the Notre Dame Hospital, Central Falls.

Dr. Lappin was a past president of the Pawtucket Medical Association, the Rhode Island Medical Society and the American Academy of Family Practitioners. He was also a former member of the Brown University Medical School faculty.

He was the husband of Anita (Bissonette) Lappin.

WILLIAM FAIN, MD

Dr. William Fain died on November 1, 1998.

Born in Providence, Dr. Fain had served on the staffs of The Miriam Hospital and Rhode Island Hospital since 1939. He was a graduate of Brown University and a 1934 graduate of Tufts Medical School. Dr. Fain was a member of Phi Beta Kappa and Sigma Si. He was a member of the American Medical Society and the Rhode Island Medical Society.

He leaves several nieces and nephews.

ROBERT W. RIEMER, MD

Dr. Robert W. Riemer died on December 3, 1998.

Born in Norwood, Massachusetts, Dr. Riemer graduated from Amherst College ('38) and Tufts Medical School ('42). He served his internship at Rhode Island Hospital and was a resident physician at Memorial Hospital.

Dr. Riemer was in the Army Medical Corps during World War II, serving in the 9th Evacuation of the Roosevelt Hospital unit. He was stationed in Europe and received the Bronze Star. After discharge, he completed his surgical residency at the Rhode Island Hospital.

In 1971 he performed the first cardiac-bypass procedure for coronary artery disease with Dr. Lester Vargas at the Rhode Island Hospital. A close friend and peer of Dr. Denton Cooley, he spent many months in Houston perfecting cardiac bypass techniques.

During his career, he was on the active and consulting cardiovascular and thoracic surgical staffs of the Rhode Island Hospital, Memorial Hospital of Rhode Island, Women & Infants, Rhode Island State Infirmary, the Veterans Administration Medical Center, the Miriam Hospital, Notre Dame Hospital, St. Anne's Hospital and Charlton Memorial Hospital. He was certified by the National Board of Medical Examiners in 1943, the American Board of Surgery in 1951 and the American College of Surgeons in 1952. Dr. Riemer was made a fellow of the American College of Chest Physicians in 1953, the Founders Group of Thoracic Surgeons in 1957 and the American Board of Angiology in 1963.

Dr. Riemer was past president of the Providence Surgical Society, the New England Chapter of the American College of Chest Physicians, the Memorial Hospital staff and the Rhode Island Thoracic Society, a former director of thoracic surgery at Rhode Island Hospital, Memorial Hospital and Zambarano Tuberculosis Sanitarium.

He was a former director of cardiovascular thoracic surgery at St. Anne's Hospital, Rhode Island Hospital and former chief of surgery at Charleton Memorial Hospital. He served as a clinical associate professor of surgery at Brown University.

Dr. Riemer was a member of the Providence Medical Society, the Rhode Island Medical Society, the Pawtucket Medical Society, the Providence Surgical Society, the Rhode Island Thoracic Society, the Massachusetts Medical Society, the Bristol County South Medical Society, Norfolk Medical Society, American College of Surgeons, American Thoracic Society, American Eastern Thoracic Society, Roosevelt Hospital Alumni and the Fall River Medical Society.

He was the husband of Gladys (Lindblom) Riemer. Besides his wife, he leaves two sons, Robert H. Riemer of Amenia, NY, and J. Michael Riemer of Seekonk; two daughters, Ann North of Manhattan, Il., and Gretchen Pace of Boston; three sisters, seven grandchildren and two great-grandchildren. He was the father of the late Air Force Lt. Richard W. Riemer.

HENRY C. MCDUFF, JR., MD

Dr. Henry C. McDuff, Jr, died on December 6, 1998.

Born in Pawtucket, he lived in Providence most of his life. Dr. McDuff graduated from Phillips Andover Academy ('34) and Dartmouth College ('38). He attended graduate school at Brown and received his medical degree from Cornell Medical College in 1943. He served in the Army Medical Corps during World War II.

He returned to Rhode Island in 1950 after he completed training in New York, and became assistant surgeon in gynecology at Rhode Island Hospital. He was appointed chief of the department in 1953 and president of the medical staff in 1963. He was a member of the staff of the former Lying-In Hospital, now Women & Infants Hospital, from 1950 until 1986. He was a consultant in gynecology at Roger Williams Hospital, Woonsocket Hospital and Newport Naval Hospital. After retiring from clinical practice in 1986, Dr. McDuff became medical director of Hospice. He retired from Hospice Care of Rhode Island in 1992.

Among his professional memberships were the Providence Medical Association, Rhode Island Medical Society, American Medical Association, Providence Surgical Society, Friday Night Medical Club, Johnny Cake Society and the Obstetrical and Gynecological Travel Club. He had been a president of the Obstetrical Society of Boston and the New England Association of Gynecological Oncologists.

Dr. McDuff was the husband of the late Charlotte (Lewis) McDuff. He leaves a son, a daughter, two grandchildren and a great grandchild.

TARAS HANUSHEVSKY, MD

Dr. Taras Hanushevsky died on December 7, 1998.

Born in Dora, West Ukraine, he lived in North Providence for 39 years.

Dr. Hanushevsky had been on the medical staff at Roger Williams and St. Joseph's Hospitals. He was a member of the American Medical Association and the Rhode Island Medical Society. He had been a president of the New England branch of the Ukrainian-American Medical Society, of which he was the founder. As a physician, he was known for his diagnostic skills and caring attitude. He was a member and former trustee of St. Michael's Ukrainian Catholic Church in Woonsocket.

Dr. Hanushevsky was the husband of Ivanna Nee (Danylovych) Hanushevsky. Besides his wife, he leaves three daughters, a son, a brother, four sisters and eight grandchildren.

WILFRED I. CARNEY, SR, MD

Dr. Wilfred Carney died on December 31, 1998.

Born in Youngstown, Ohio, he lived in Providence most of his life. Dr. Carney attended St. Vincent's College in Latrobe, PA, and received his medical degree from Jefferson Medical College, Philadelphia. Dr. Carney's surgical training at Rhode Island Hospital and Truesdale Hospital in Fall River was interrupted in 1940, when he joined the Army. He was stationed at Fort Benning, Georgia, and Opelika, Alabama. As a lieutenant colonel, he managed a 1000-bed hospital in Panama for more than two years.

After World War II, he completed his surgical training at the Rhode Island Hospital. He was chief of first and second surgical services at Rhode Island Hospital. He was an associate clinical professor of surgery at Brown University Medical School. Among his professional memberships were the Providence Surgical Society, Rhode Island Medical Society, New England Surgical Society, American Medical Association and the American College of Surgeons.

He was the husband of Mary (Truesdale) Carney. Besides his wife, he leaves four sons, Dr. Wilfred I. Carney, Jr, Stephen T. Carney, Dr. Martin J. Carney and David T. Carney; four daughters, Jane C. Forbes, Dr. Nancy D. Carney, Dr. Mary C. Cillon and Diana C. Caty; 29 grandchildren and 2 great-grandsons. He was the father of the late Sarah E. Carney.

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Ninety Years Ago

[March, 1909]

L.C. Kingman, MD, gave the history of "Blood Transfusion" from the first attempt to the present, which he conceded was "still in some respects in the 'experimental stage.'" He praised the technic of Cleveland's Dr. Crile and discussed the use of a magnesium ring (invented by Paar in 1900) for permanent anastomosis between the donor's artery and the recipient's vein. Kingman cautioned: "[transfusion] does not supersede saline infusion. ... Transfusion takes up the work where infusion leaves off."

Donald Churchill, MD, presented "A Report of Three Cases of Purulent Peritonitis." This surgeon urged "surgical neglect," or "the courage to do as little as possible." He recognized the confinement of pus, not pus itself, as the cause of illness: "Pus confined anywhere in the human body makes itself known and feared, not because it is pus, but because it is confined. Because it is confined, its toxines are absorbed." He cut only enough to allow the pus to drain, relying on gravity: "An opportunity for the pus to get out is what we should aim to accomplish. It will come out of itself if you give it a fair chance, and you cannot hurry it one bit by trying to make it go faster."

Twenty Five Years Ago

[March, 1974]

Pierre Galletti, MD, PhD, Vice President (Biology and Medicine), Brown University, wrote the lead editorial, "The Cost of Medical Education; and Who Should Pay for It." An AAMC survey had pegged the cost of one year of medical education at $16,300-$26,400 (including faculty research time). In 1971, when Congress passed the Health Manpower Act, Congress sought to increase the supply of physicians; and the government subsidized much of the costs. In 1974, Galletti reported, "..the goal of a sufficient number of physicians seems to be at hand." The current Congress was discussing the distribution of physicians, and the responsibility for payment. "There seems to be a concern ... that in the near future medical students will be asked to pay a greater share of medical education costs through tuition, and that loan programs will largely replace scholarship and grants."

Fifty Years Ago

[March, 1949]

In "X-Ray Treatment of Malignant Lymphoma," Emanuel W. Benjamin, MD, discussed the 23 cases treated (with topical x-ray treatment) at Memorial Hospital's tumor clinic since its inception. While patients were not cured, Dr. Benjamin noted "encouraging" results "as regards prolongation of life and alleviation of symptoms."

F. Ronchese, MD, and Bencil L. Schiff, MD, presented a study of "Diabetic Lipoidic Necrobiotic Dermatitis." Patients who had the specific skin lesions on one or both legs, usually below the knee, often had diabetes as well: "...in more than 87.2% of this skin disorder, diabetes was present (severe or moderately severe)." Those patients without diabetes mellitus, moreover, may have been in a prodromal stage for diabetes. In some cases, lesions predated the diabetes by as many as 8 years.

The Journal reprinted the AMA's "Plan of Battle: An Outline of Strategy and Policies in the Campaign Against Compulsory Health Insurance," part of the AMA's National Education Campaign to defeat legislation for compulsory health insurance, supporting instead a drive for more voluntary health insurance. The authors discounted any public enthusiasm for compulsory insurance: "...the major demand for compulsory health insurance in the United States is an artificial, trumped-up demand, generated by social bureaucrats."

The Rhode Island Medical Society's House of Delegates, arguing against a" primarily punitive" proposed state drug bill, supported alternative legislation that would emphasize rehabilitation and treatment. "We look upon the abuser generally as one to be helped with his problems rather than being punished for his transgressions...We firmly believe that society will best be served if people are rehabilitated instead of punished."

Charles H. Mandell, MD, introduced a series of papers on nuclear medicine, describing the procedures as "safe and ...[with] relatively low radiation exposure."

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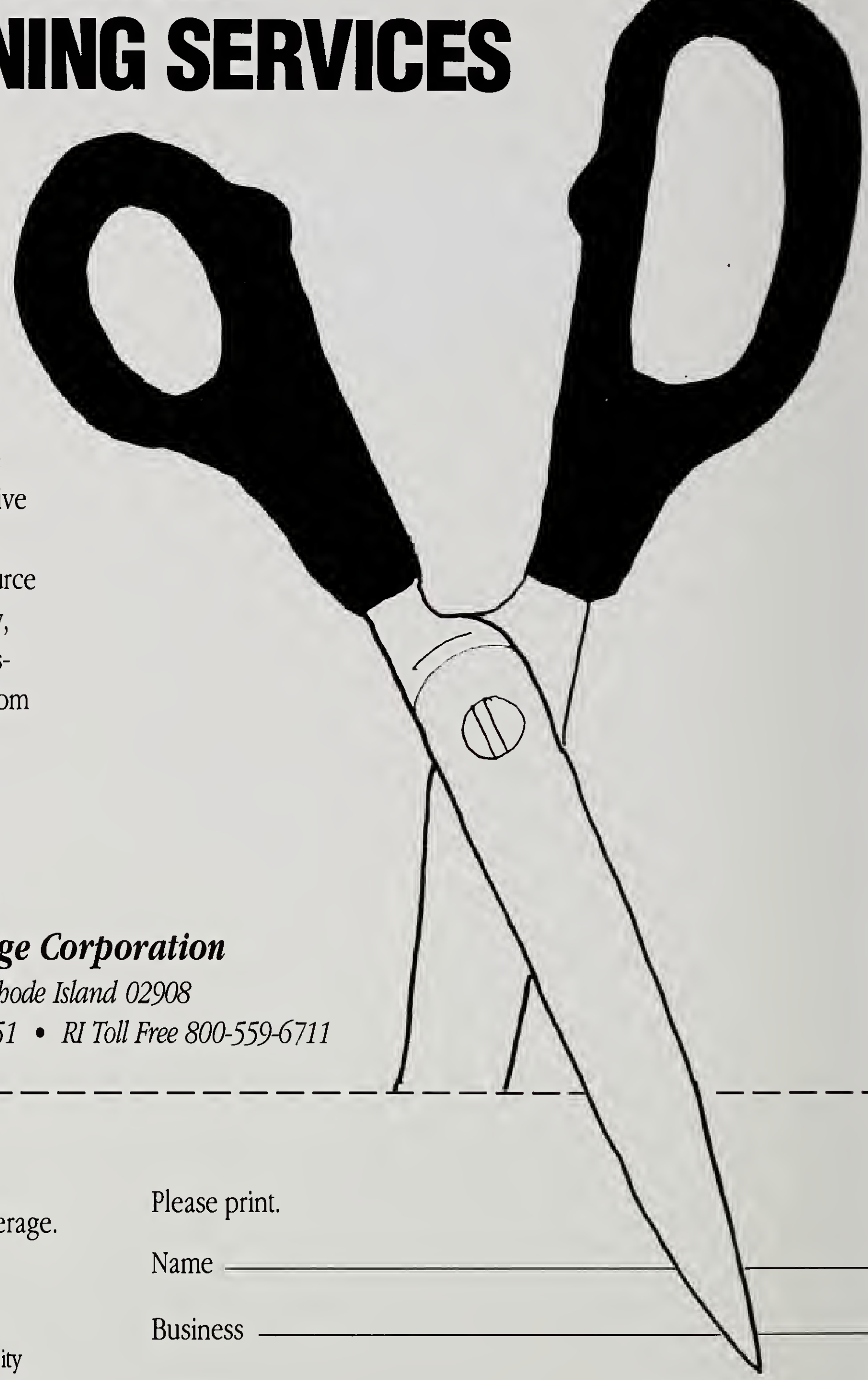

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COMMENTARIES

Cure after Cure after Cure

Sometime around the beginning of the new year I learned that angina had been conquered. Radio news and newspapers blasted the latest medical advance (I don't watch TV news but I assume it was there too) that a direct injection of an endothelial growth factor into the heart produced angiogenesis and angina relief. Last year a prominent researcher cured cancer, at least in mice, so spectacularly that strong attempts were made to jump start to human trials. This year those spectacular results weren't reproducible. I've seen Parkinson's disease cured several times in the past decade. And while claims of Alzheimer's disease cures haven't been made, the various major breakthroughs in the news haven't led to noticeably better treatment.

These "cures" remind me of an old joke I learned in medical school. When a clinician has seen one case, he speaks of, "my experience." With two cases he speaks of, "my series." With three cases it becomes, "in case after case after case."

Our patients are seeing "cure after cure after cure" in the news while we often have little new to offer in the office. Even when the patients themselves have been beaten down by repeated disappointments, their well-meaning friends, neighbors, and relatives push them to grasp at the latest straw. "Ask your doctor about injecting (choose one) human fetal brain tissue, stem cells, pig fetal brain cells, gene engineered fibroblasts, growth factors."

And while I blame the news media for this, (we) researchers are partly to blame as well. We all want our 15 minutes of fame. We want our patients to see that we are "famous." We use this public exposure to deal with our chairs and administrators in the usual jockeying for space, time and money. The journals can't get enough attention either. The *New England Journal* muscled its way to the media forefront a few years ago and then developed a rule that forbids public mention of results until publication. Violation of this rule means withdrawal of publication. This and the other fame-seeking journals mail their issues to the news media before sending them to their subscribers. And the specialty societies aren't much different. They have schedules for "media worthy" research at their annual meetings. In addition, a lot of research is either performed directly by private companies or is sponsored by them; and well-timed research "breakthroughs" help manipulate stock prices.

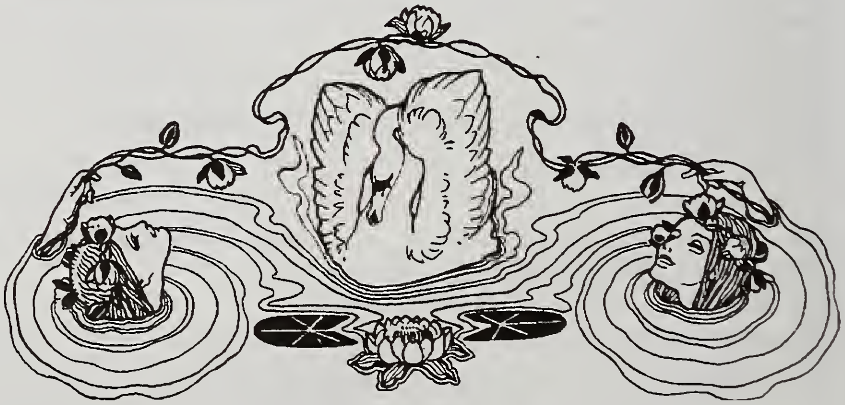
How can this be controlled? I think by having more responsible editorial oversight at the news media. Patients need to have hope. They need to hear that researchers are slogging away at their particular illness, that advances are being made. But they shouldn't be hit over the head with two inch headlines that the end is in sight. I tell my patients that medical research is analogous to the European explorers' search for the origin of the Nile River. Every day they sketched out more of the map but they never knew what they would find when they climbed the next hill. I think we all need some restraint and that the media needs to put their reports into context.

As I was writing this, I noted that high fiber diets no longer prevent colon cancer and that rheumatoid arthritis can be easily controlled. The map is, perhaps, now more detailed and what was presumed is now (possibly) discredited.

Conclusion 1: Discretion is a virtue we should exercise more strongly in our medical publicity.

Conclusion 2: "New remedies should be used quickly," suggested Osler, " while they still work."

– Joseph H. Friedman, MD



Staggering Lambs, Stricken Mink, and Mad Cows

Eighteenth Century Scottish shepherds noticed an inexplicable change in some of their sheep. Affected ones staggered, displayed limb tremors, and weight loss. They could easily be spotted from afar by their characteristic tendency to rub their bodies against fence posts; and this compulsive scratching, often with the scraping away of wool, prompted the herders to call the disease *scrapie.* Once begun, the disease inexorably led to death, generally in months. In 1936 the disease was shown to be infectious. And because of the unusual chronicity of the disease, it was thought to be caused by a "slow virus." Currently, this economically destructive disease is worldwide [except for Australia] with many nations embargoing sheep importation and destroying any sheep showing early signs of scrapie.

In the years following World War II, missionaries and government agents documented a heretofore undescribed disease affecting aboriginal inhabitants of the remote highlands of Papua New Guinea. The disorder seemed to be confined to a Stone Age tribe called the Fore people numbering about 14,000 natives. The brain disease began slowly, but once begun led invariably to death within months or years. The victim, almost always an adult woman or a child of either sex, developed an unsteady gait, uncontrollable tremors, inappropriate behavior, disturbed speech, weight loss, progressive dementia and ultimately, total helplessness. Natives called it ***kuru,*** a local word meaning trembling and ascribed it to sorcery. Scientists eventually demonstrated that kuru brain tissue, when injected into healthy animals, produced a similar disease. However, the incubation period [the interval between introduction of the tissue until the time when pertinent symptoms arise] was measured, not in days but in years. A larger puzzle remained. How did this uniformly fatal disease spread from person to person ? Medical anthropologists, living with the Fore people, finally supplied the answer. Ritual cannibalism was still practiced by the women as a means of honoring their dead; and they and their children regularly consumed human tissue. When, in the 1950s, the authorities banned cannibalism the disease gradually disappeared.

In seeking to understand kuru, pathologists noticed that the changes in the brain tissue of victims closely resembled those encountered in humans dying of a rare form of dementia called Creutzfeldt-Jakob disease [CJD]. And shortly thereafter, in 1959, a veterinary pathologist demonstrated that these destructive changes [showing, amongst other unique features, a microscopic sponginess to the brain tissues] also characterized the brains of scrapie-affected sheep. By 1968, CJD brain tissue, when injected into experimental animals, created a replica of CJD but only after a lengthy incubation interval. A series of human tragedies in the 1960s then confirmed the infectious nature of CJD. Corneal tissue, salvaged at autopsy, has been used routinely in restoring vision in certain forms of blindness. In those very rare cases in which the donor had been a patient with CJD, the transplant recipient, years later, also developed CJD. [There are now careful criteria for the harvesting of post-mortem corneas, and the risk of CJD in transplant recipients has disappeared.]

Wisconsin mink farmers faced their own perils in the 1960s when a mysterious wasting disease occasionally wiped out their caged colonies. Only after careful microscopic analysis of the brain tissue of affected minks was the resemblance to scrapie, kuru and CJD finally affirmed.

Gradually, then, a new category of disease was recognized, with the following common characteristics: They were chronic, invariably fatal, and confined to the brain; incubation intervals were prolonged; each could be successfully transferred to experimental animals; and the customary landmarks of infectious disease, namely, fever, inflammation and antibody response, were notably absent. The diseases were collectively called the transmissible spongioform encephalopathies [TSE].

As more research was undertaken, the fuller, and more terrifying, dimensions of the TSEs gradually emerged. Similar renegade diseases were encountered in feral populations including western elk and deer. In terms of economic impact, however, the worst was yet to come. In 1985 a strange disease appeared in England's herds of cattle and dairy farms. Staggering gait and bizarre behavior caused many to call this mad cow disease. By 1991 over one million cattle were manifestly sickened and had to be destroyed. Delays in recognizing the public health ramifications allowed much of the tainted British beef to be consumed by humans. A statutory ban on feeding slaughter house-derived protein to domesticated animals had finally been imposed in 1988, but by 1994 there was a tragic outbreak of CJD amongst Britons.

The TSEs, while infectious, are not contagious in the conventional sense. The diseases are not transmitted by the customary pathways of contagion, namely, by air or by touch. No physician or nurse tending patients with kuru has ever come down with the disease. How then do these infective TSE agents move from the infected to the uninfected? What, scientists asked, do mad cows, ataxic mink, staggering sheep and trembling Fore natives have in common ?

In each case, the infective agent was probably consumed with food. In the case of kuru, by ritual cannibalism; in the case of the mink, by feeding them the carcasses of sheep, many probably dying of scrapie; in the case of the British cattle, by a supplemental feed derived from slaughter house remnants. Only the CJD cases remain unexplained.

In 1997, Kentucky physicians found a cluster of CJD cases in people who ate a local delicacy called burgoo [stewed wild squirrel brain], adding still another unanticipated hazard to the TSE category.

Many questions remain: What is the nature of this strange organism or agent ? Most studies indicate that it contains no nucleic acid [thus making it the only known pathogenic organism without DNA or RNA]. One American scientist, Dr. Stanley Prusiner, working on the problem for decades, has concluded that we are confronting a new disease agent distinguishable from and more rudimentary than the viruses. He calls them prions. No therapy has been shown to have any affect upon the course of these diseases. Public health measures, therefore, have been directed toward imposing preventive barriers such as the prohibition of cannibalism in New Guinea, the abolition of animal byproducts for the routine feeding of livestock or the creating of rigorous standards when determining the suitability of human tissue for transplantation.

The immediate need, then, is for further preventive measures and more basic research. And when weekly publications tell of the imminent conquest of all contagious disease, there is also a need for skepticism, tempered with humility. Those innocents who feel safely insulated from the highlands of New Guinea or the rainforests of Africa will not be treated kindly by history.

– Stanley M. Aronson, MD

Third Annual Research Symposium on Mental Health Sciences Abstracts: Introduction

Martin B. Keller, MD

The Brown University School of Medicine Department of Psychiatry and Human Behavior (DPHB) was pleased to present The Third Annual Research Symposium on Mental Health Sciences on December 1, 1998. The DPHB was honored to host guest speaker, Enoch Gordis, MD, Director of the National Institute on Alcohol Abuse and Alcoholism (NIAAA), National Institutes of Health. Brown University faculty presenters at the symposium were Henrietta Leonard, MD, Richard Longabaugh, EdD, and Rena Wing, PhD. The symposium also featured inspiring introductory remarks by Donald J. Marsh, MD, Dean of Medicine and Biological Sciences at Brown University. Faculty, post-doctoral fellows, residents, psychology interns and medical students from Brown University presented over 100 research abstracts and posters. Overall, 200 members of the Brown University and Rhode Island health care communities participated in this unique forum for the exchange of new research and ideas in the field of psychiatric and behavioral science.

In his presentation, Dr. Gordis outlined the history and progress of psychiatric research in the assessment and treatment of alcoholism. He suggested that the myriad of scientific advances made by researchers in addiction medicine was already improving the treatment of alcoholism and helping to focus the efforts of biological and behavioral researchers to make treatments more efficacious.

Dr. Gordis's presence was particularly welcome at the research symposium as several pioneering programs in research on alcoholism being conducted by DPHB faculty are located at the Brown University Center for Alcohol and Addiction Studies (CAAS), The VA Medical Center and The Division of Behavioral and Preventive Medicine at Miriam Hospital. One of the most exciting programs in the CAAS is the Alcohol Treatment and Early Intervention Research Training, a T32 NIAAA-funded postdoctoral training grant that prepares professionals to conduct high quality research in early intervention and treatment of alcohol abuse and alcohol dependence. Another training program, which is a collaboration of the CAAS and VA Medical Center, is the Fellowship Program in Substance Abuse Training, the purpose of which is to train physicians for academic careers in substance abuse treatment and prevention. I am pleased with the success of these and all Department programs that address the critical issues surrounding alcohol and substance abuse treatment. The collaborative efforts of our faculty and the affiliated institutions of the Brown University School of Medicine are a model of how the DPHB continues to strive for excellence, both in addiction medicine and psychiatric and behavioral medicine in general.

The success of the Third Annual Research Symposium on Mental Health Sciences and the seminal findings reported by our faculty presenters, testify to the need to continue to pursue research that helps us to understand the etiology, psychopathology and treatment of mental disorders which enables the health care community to better understand these conditions and available treatments.

Martin B. Keller, MD, is the Mary E. Zucker Professor and Chairman of the Department of Psychiatry and Human Behavior, Brown University School of Medicine.

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Advances in Research on Alcoholism and What They Promise for Future Treatment and Prevention

Enoch Gordis, MD

OF THE MAJOR MEDICAL DISCIPLINES, psychiatry has the longest involvement in treating alcoholism. The current explosion of research on the biological and behavioral causes and consequences of alcohol-related problems promises to make this task both easier and more effective. The natural history of alcoholism progresses from alcohol use, to a high priority for it, to dependence with issues of exposure on one end and craving, tolerance, and withdrawal at the other. The features associated with each stage along the continuum to alcohol dependence, including genetic vulnerability, environment pressures, the increasingly high priority for alcohol in an individual's life, reasons and the development of the hallmark symptoms of alcohol dependence (craving, tolerance, physical withdrawal and craving) are what alcohol scientists are trying to explain.

Today's alcohol researchers are working on the cutting edge of technology. Yet, just 30 years ago, science, by and large, did not consider alcohol-related problems a viable topic of inquiry. How did we get from there to here? Several major conceptual advances in science in general and in the alcohol scientific field in particular have helped to move the study of alcohol dependence forward. One such advance has been the clear demonstration that a portion of the vulnerability to alcoholism is inherited. Although we still do not know precisely what is inherited or whether what is inherited is specific to alcohol, this conceptual advance has led to an explosion in human and animal genetic work that promises to lead to the development of new treatments and better focussed prevention efforts for those most vulnerable to developing alcohol dependence. Already scientists participating in the National Institute on Alcohol Abuse and Alcoholism (NIAAA)-supported Collaborative Project on the Genetics of Alcoholism and in NIAAA's own laboratories have identified a number of chromosomal "hot spots" related to alcoholism. These and other scientists are now looking for the genes that are active in the development of alcoholism.

A second conceptual advance is the application of neuroscience to understanding drinking and the phenomenon of addiction. Alcohol neuroscientists are learning much about how alcohol affects various neural systems to produce alcohol dependence. New imaging techniques have permitted neuroscientists to study alcohol's effects on the brain in ways not even possible just a decade ago. This conceptual advance, among other things, has led to the development of new pharmacotherapies for alcoholism treatment, such as naltrexone and acamprosate.

The acceptance of the study of "mental processes" in the study of alcohol's actions is a third advance. Understanding that certain prior experiences with alcohol affect reactions to it has led to two important areas of science: the issue of expectancies, where some of the actions of alcohol are produced because they are expected, not because of the drug's pharmacology; and the fundamental issue of craving, specifically, whether craving exists intrinsically or whether it is dependent on cues from the environment.

Another important advance has been in our understanding of how alcohol damages the body, including new understanding of withdrawal and how to safely detoxify patients, increased understanding of the mechanisms involved in alcoholic liver damage, and how alcohol damages the fetus. We also have a much better understanding of alcohol's protective effects against coronary artery disease and possible protection of postmenopausal women against osteoporosis.

A fifth advance, one that has done much to establish the credibility of alcoholism treatment within the medical establishment, has been the application of all the techniques of the classic clinical trial; i.e., randomization, appropriate controls, blinding, power calculations, defined and objectively measured outcomes, and alcoholism therapies. We are not only testing new therapies, but also older treatments that have been used over time but never subjected to scientific scrutiny. Related to this is important research on alcohol treatment in current health care systems, and how alcoholism treatment services are designed, accessed, and paid for. This type of data has become increasingly necessary in today's managed care environment.

The last conceptual advance is the now demonstrated fact that it is possible to do controlled trials in prevention, and that research on social and regulatory policies can be applied in policy formulation to decrease or prevent risk from alcohol-related problems. This has been demonstrated most dramatically in the significant number of young lives saved as a result of a national policy to raise the minimum legal drinking age to 21. As we begin the new millenium, these advances in alcohol research will bring not only the excitement of scientific disocvery, but the fulfillment of its promise to prevent and effectively treat alcohol-related problems.

Enoch Gordis, MD, is Director of the National Institute on Alcohol Abuse and Alcoholism (NIAAA), National Institutes of Health.

Matching Alcohol Treatment to Patient Social Support and the Effects of Participation in Alcoholics Anonymous

Richard Longabaugh, EdD

AT PRESENT, SCIENTISTS HAVE LITTLE IDEA why a treatment for alcohol dependence works when it does. Tests to uncover the "active ingredients" of treatment interventions are just getting under way. The study presented in this talk investigates the role of social support on treatment outcome.

We test the hypothesis that a Twelve Step treatment will be more effective than a motivational intervention aimed solely at mobilizing the patient's own resources only when the patient has a pre-treatment social network that is supportive of drinking. This finding emerges clearly three years after treatment, but not before. To account for this emergent treatment effect, we test the hypothesis that involvement in Alcoholics Anonymous (AA) is an active ingredient.

We conclude that, especially in the current climate of managed care, treatment providers must broaden the focus of their interventions to include factors in the patient's natural environment. Use of AA as an adjunct treatment should be considered for all alcohol dependent patients, but it is most helpful for those who come to treatment with social networks supportive of the patient's drinking.

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Childhood Onset Obsessive Compulsive Disorder: Is There a Unique Subtype?

Henrietta Leonard, MD

ONLY TEN YEARS AGO, THE PERCEPTION EXISTED that obsessive compulsive disorder (OCD) was rare in childhood. Current studies suggest that one in 200 young people suffers from OCD. The fact that one-third of adults with OCD develop the disorder during childhood or adolescence indicates that it may lead to substantial adult morbidity.

Research on the phenomenology, neurobiology and the psychopharmacologic treatment of OCD and Tourette's Syndrome (TS) has led to the current conceptualization of these disorders as neuropsychiatrically-based disorders with regional brain dysfunction. Despite extensive investigations, the gene(s) has not been localized. Focus has shifted to determine whether there are identifiable phenotypes and to what extent nongenetic factors may mediate the expression of the illness.

Recently, a group of children with OCD and/or tic disorders associated with group A beta-hemolytic streptococcal (GABHS) infections were studied, and subsequent systematic clinical evaluations were performed on 50 children who met all of the following criteria: presence of OCD and/or tic disorder; prepubertal symptom onset, episodic course of symptom severity; association with GABHS infections; and association with neurological abnormalities. Their course was characterized by relapsing-remitting symptom pattern with significant psychiatric comorbidity accompanying the exacerbations, including emotional lability, separation anxiety, cognitive deficits, oppositional behavior, and motoric hyperactivity. Working diagnostic criteria appear to accurately characterize a homogeneous patient group in which symptom exacerbations are triggered by GABHS infections (described as PANDAS, Pediatric Autoimmune Neuropsychiatric Disorders Associated with Strep). Controlled antibiotic and immunomodulatory trials were presented. The identification of such a subgroup will allow for testing of models of pathogenesis, as well as the development of novel treatment and prevention strategies.

Henrietta Leonard, MD, is Professor of Psychiatry and Human Behavior, Brown University School of Medicine. She is also Director of Training for the Child and Adolescent Psychiatry and Triple Board Programs.

Behavioral Strategies to Improve Long-term Weight Loss and Maintenance

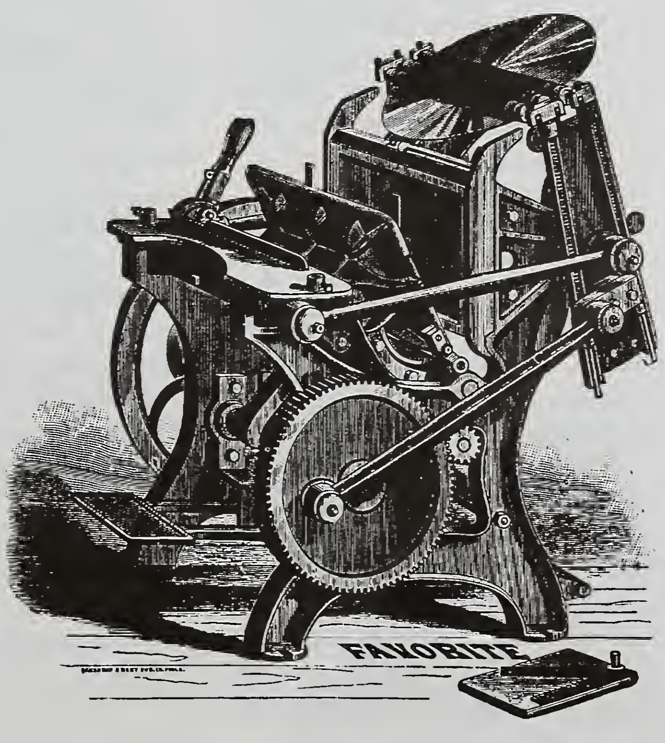
Rena R. Wing, PhD

BEHAVIOR THERAPY HAS BECOME QUITE successful in producing initial weight loss. On average, patients in behavioral programs lose 20 lb. (10% of their initial weight) within the first 6 months of treatment. Maintenance of weight loss is, however, still a problem. When reexamined one year after treatment, participants have typically regained one-third of their initial weight loss. Several new approaches have been utilized to improve these long-term results. These include: a) increasing initial weight loss through stricter diet regimens; b) increasing duration of treatment; c) focusing more on the skills required during maintenance; d) using stronger behavioral approaches to increase adherence to diet and exercise; and e) changing the prescription for diet or exercise. The latter two approaches were described in detail during the talk.

Dr. Rena Wing and her colleague, Dr. Robert Jeffery in Minnesota, have utilized stronger behavioral approaches to change the antecedents and consequences for weight-related behaviors. For example, participants in weight loss programs are typically taught to purchase more low calorie foods and decrease the number of high calorie foods. However, these investigators found that participants had better dietary adherence if, rather than being instructed to make changes in their food purchases, they were actually given the foods they should eat in appropriate portion sizes or at least given suggested meal plans that modeled specific ways to consume a low calorie, low fat diet. Likewise in the area of exercise, these investigators found that participation at supervised exercise sessions could be improved by providing personal trainers or "coaches" who called participants and walked with them at these exercise sessions or by providing small financial incentives for exercise attendance. However, although these techniques improved exercise adherence, overall self-reported physical activity was not increased nor was weight loss improved. Interestingly, the top quartile of subjects in this study, who reported averaging over 2500 kcal/week in exercise, had excellent long-term weight loss. This level of exercise, which contrasts sharply with the 1000 kcal/week goal typically used in weight loss programs, has led to the question of what is the appropriate exercise goal to recommend for long-term weight loss.

A higher exercise level also emerged as a correlate of long-term weight loss maintenance in another study Dr. Wing presented. Dr. Wing and Dr. James Hill, of the University of Colorado, have established a registry of individuals who have lost at least 30 pounds and kept it off for at least one year. The National Weight Control Registry now includes over 2500 individuals who have lost, on average, 60 pounds and kept it off over 6 years. This is the largest cohort of successful weight losers. These individuals report that they continue to consume a low calorie, low fat diet (<24% of calories from fat) and expend, on average, 2800 kcal/week in physical activity. This figure, and the types of exercise they report performing, are quite similar to the most successful participants in the study described above. Other interesting findings reported from the Registry indicate that almost half of these successful weight losers lost weight on their own (with no professional help), most weighed themselves at least once a day, and most report that this effort at weight loss (versus earlier, less successful attempts) involved more commitment to behavior change, greater exercise, and a stricter diet. Hopefully, studies such as these and new strategies that emerge from them will help produce more successful long-term weight control programs.

Rena R. Wing, PhD, is Professor of Psychiatry and Human Behavior, Brown University School of Medicine. She is also Director of Diabetes and Obesity Research in the Division of Behavioral and Preventive Medicine at Miriam Hospital.



The Lived Experience of Chemotherapy for Women with Premenopausal Breast Cancer

Carol Jacques, RN, NP

In the U.S., one woman in 9 over the course of her lifetime will be diagnosed with breast cancer. It is the leading cause of death in women aged 15-54, and the second for women aged 55-74. Chemotherapy has become the standard treatment regardless of the invasiveness or size of the tumor. Preliminary results show it to be successful in treating breast cancer; yet the powerful drugs result in many physical and emotional problems for the patient. Young women, healthy except for a tumor which caused no symptoms, are subjected to chemicals which cause severe side-effects, and sometimes death.

In order to understand the effects of chemotherapy on premenopausal patients with breast cancer, a phenomenological study was conducted. Each woman was asked: "Please describe what it was like for you to have received chemotherapy." Each participant was allowed to talk uninterrupted. When she stopped, her last statement was repeated back to her, and she was allowed to continue talking. When she had nothing left to say, the interview ended. The data was analyzed according to Colaizzi's 6-step method for Phenomenological Research.[1] From the transcripts, significant statements were grouped into themes. Two of the 4 participants read the findings to validate the researchers' findings. Two refused, due to the emotional content of the topic. Twenty-five women who had had chemotherapy for breast cancer read the analyzed data and agreed with the results.

The study was conducted in Southern New England. The sample size was small (4); ages ranged from 33 to 43 years at the time of chemotherapy. At the time of the interviews, the women were cancer free, and had had chemotherapy in the previous three years. Three women had a family history of cancer: One woman's mother had died at age 36 of breast cancer; another's was alive and well aftter breast cancer in her fifties; the mother of a third had died from brain cancer (primary site).

Twelve themes were identified - described eloquently in the patients' own words.

Theme 1. **Lack of preparation.**

Although chemotherapy is standard treatment for premenopausal women with breast cancer, these patients were unprepared for it. "..... the chemo thing didn't hit me until I was there and they were putting it into the porta-cath and pumping that pink stuff in." (Ten years ago, physicians were largely responsible for explaining the mechanics, and side-effects, of chemotherapy; today that task belongs to nurses. This study did not examine the extent of pre-treatment nursing counselling, or the information imparted. Whatever the imput on the nurses' side, though, little information reached these patients.)

Theme 2. **Abdication of family caretaker role.**

In order to concentrate on herself during chemotherapy, a woman must relinquish her responsibilities as primary provider of emotional and physical support for her family. Culturally, women's roles have been as nurturers. For some women, abdication of this role is the most difficult part of the illness. As difficult as it is to relinquish this role of nurturer, she arranges a support system for her family. " I knew what I had to do for my own survival. And what I had to to do was look into myself and save all of my strength for myself, because I was scared, like anybody else, and this time I was going to be selfish."

Theme 3. **Importance of patient empowerment.**

Taking an active role in treatment minimizes the anxiety of the unknown, and gives a feeling of self-worth. Asking questions of the doctors and nurses, reading books on chemotherapy and breast cancer, and maintaining a positive attitude helped reduce the stress of treatment. "If you have control over your illness and your treatment, if you get involved, if you find out what is going on...then I think that maybe more positive things can happen. But I think that if you leave it in somebody else's hands, then you've given up."

Theme 4. **Need for supportive network**

To keep a positive attitude through the treatments, patients discussed problems with close family and friends, and avoid those people who are upsetting. "What I cannot tolerate is people who feel bad for themselves. They feel bad, and they don't look at what they have. If they have their health, they are the luckiest people in the world." "Emotionally, it's hell."

Theme 5. **The escalation of misery.**

Side effects increased in severity with each treatment, along with the threat of infection from a suppressed immune system..."The fourth time was terrible. I mean, after the third time, I begged my husband, 'Please don't make me go back again!' but the fourth time was really bad. But I took solace in the fact that it was over."

Theme 6. **Hair loss.**

Hair loss was the most emotionally traumatic experience; however, it motivated the women to regain control of their bodies and their lives. "I would be sitting at the table and I would hear it dropping. It would make little pings on the table: ping, ping,

ping, ping. So, eventually, it started getting thin, and then...[in the] shower, it was all sticking to me like seaweed, and it was really horrifying. And it looked so awful. I looked sick...I finally just stood over the wastebasket and just kept pulling it out."

Theme 7. A physical edginess.

The chemotherapy brought on a physical edginess, which resulted in uncontrollable emotional outbursts. One woman took off her wig and screamed at her husband, "Look at me! Look at this!"; she laid on the couch and cried herself to sleep. Another became "very, very angry. And all that came out through the chemo. Before the chemo it didn't come out, and after the chemo, it didn't come out. Because I was always on edge. Physically, it made me very fatigued. I had no patience. If somebody dropped something on the floor, I would jump. I was always on edge."

Theme 8. Social isolation.

Side effects from chemotherapy were socially isolating and emotionally draining. A very serious side effect was a low WBC (white blood count). Women understood that what was a simple cold or flu to others could be life-threatening to them. Only 1 woman escaped seeing the inside of a hospital as a patient.

Theme 9. Children's presence.

Most children did not ask how their mother's chemo was going - they judged it for themselves. "... I would come home after chemo, flop on the sofa in the living room, and when [my son] came home from school, he woud sit next to me in the chair, do his homework there. Even when I was sleeping, he'd be there. If I needed something, he jumped up to get it. That happened for those 2 days, the 2 chemical days, he would sit right there, not saying much."

Theme 10. Post-treatment disorientation.

When chemotherapy ended, there was relief, but no elation, followed by a lack of direction and a heightened sense of personal mortality. "And after the radiation and it was all over...I sat down and said, 'What do I do now?'....It's like I got off the bus, stayed for awhile and got back on the bus and didn't really know where I was going." "I don't know how old I'll be when I die, but it's just actually helped me realize my mortality That forever isn't a real long time." "Sometimes I don't know where I'm going. While I was getting the treatment, I felt at least I'm doing something. The last treatment, I was feeling, 'This is the last time I'm doing something.' And then you're on your own...."

Theme 11. Post-treatment health.

Problems whch arose post-chemotherapy were attributed to the drugs. Two women experienced health problems. "And everyone I ask says, 'Oh, it's completely unrelated to the chemotherapy.' I have to believe that. It doesn't really matter at this point, because no matter what caused it, they treat it in the same way, but I wonder if I had not had the chemo, maybe this wouldn't have happened." Another woman half-jokingly said, "I think I have brain damage from the chemo! Oh, I don't know, maybe not brain damage, but it's definitely done something to my mind."

Theme 12. Post-treatment resolve.

From the chemotherapy experience, the women grew emotionally and spiritually, realizing that if necessary, they would do it again. They had overcome their fears not only of cancer, but of the grueling treatments. "I always wondered what it would be like if somebody called me up and said, 'You have cancer.' And now I know, I know I can do it and I can make it...I found out that I'm a survivor." "It's an unfortunate thing, but on the good side it's fortunate becaue I think it makes you a better person. It makes you more appreciative of things that you probably always take for granted and never realized that you took them for granted, and you're closer to your family. So, there are positive sides to being sick."

This paper was abstracted from the author's master's thesis in the Department of Nursing, University of Rhode Island ('96).

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Psychosocial Aspects of Gynecologic Crohn's Disease

Silvana Ribaudo, BA, and Edward Feller, MD

Because of its tendency to be transmural and to fistulize, Crohn's disease (CD) may involve the perineum or gynecologic organs. In addition to medical and surgical problems, the psycho-sexual burden of this potentially debilitating and chronic inflammatory disorder is common and not amply highlighted in standard reference works (no mention in a recent encyclopedic "textbook of women's health," for example). Phycicians caring for women should be aware of the protean gynecologic manifestations of Crohn's disease and their effect on lifestyle and emotional well-being as demonstrated by the following hypothetical clinical vignettes.

A 31 year-old woman develops a rectovaginal fistula with fecal vaginal drainage. She is sullen, afraid of the possibility of incontinent "accidents."

Vaginal and perineal fistulas are reported in up to 20% of women with CD. The diagnosis may be obvious, but potentially misappreciated if the drainage is mucoid, not feculent or a fistula is the presenting feature of Crohn's. Since these lesions are commonly chronic, major ramifications on psychosocial functioning may occur.

A 19 year-old woman has undergone a total colectomy with ileostomy for Crohn's colitis. Her body feels alien and disfigured.

Some women report disinterest in sex or fear of relationships because of the potentially embarassing reality of an ileostomy. Data also suggest that sexual partners may also experience decreased sexual interest. Conversely, after total colectomy, some patients report improved sexual functioning as a result of enhanced sense of well-being and amelioration of pain, bowel dysfunction, and the debilitating effects of chronic disease.

A 41 year-old woman has painful unilateral vulvar hypertrophy .

Crohn's disease may involve the vulva by metastatic ulcerations, erosions, chronic inflammation, fistulization from colonic disease or extension of a peri-rectal abscess. Vulvar hypertrophy may be the sole site of pyoderma gangenosum complicating Crohn's. The resulting painful lesions, potentially confused with genital herpes, tuberculosis, actinomycosis, or lymphogranuloma venereum, may interfere with body image and emotional health.

A 32 year-old woman has severe perineal CD. Her daily 3-mile run has become intolerable.

Multiple abscesses, fistulas, ulcerations can occur causing severe rectal pain, dyspareunia, and fecal incontinence. These symptoms may complicate pre-existing mood disorders or result in psychologic distress, presenting clinically as depression, anhedonia, anorexia, or insomnia.

A 54 year-old woman has frequently disabling abdominal pain and multiple episodes of diarrhea despite medication and prior surgical resection for CD. The demands of a job and the pleasure of dining out with friends are impossible.

The malnourished, chronically ill patient with persisting disabling symptoms is prone to problems of self-esteem, loss of interest or desire to socialize and major disruption of daily living. Physicians need to ask patients how chronic disease affects psychological and social well-being.

Abbreviations Used:

CD	Crohn's disease

Discussion

The psychosocial toll of chronic inflammatory bowel disease has diverse manifestations which may be ignored by both patient and physician in the context of complex medical and surgical management decisions. Documented cases exist in which women neglect to disclose fecal vaginal discharge from fistulas, painful vulvar lesions or the torment of severe perianal disease due to fear of possible disfiguring surgery or embarassment. Issues of sexuality are important to discuss. Perineal pain with intercourse may occur in patients with perieal fistulas or abscesses. Patients may complain of a sensation of rectal pressure with intercourse or are inhibited because of fear of rectal incontinence.

Clinicians must be alert to the psycho-social burden of chronic disease. In a large Cleveland Clinic study of 692 women who had undergone surgery for inflammatory bowel disease, loss of pleasure or desire for sex was reported by 50% of repondents, symptomatic vaginal discharge by 40%, and abdominal pain with sexual intercourse by 50%. The majority also reported menstrual abnormalities and a much higher than expected frequency of infertility and gynecologic surgery.[1] Physicians caring for patients with inflammatory bowel disease should be aware of the diverse gynecologic manifestations and be sensitive to the associated psycho-sexual morbidity. The comforting power and practical importance of the doctor-patient alliance must be remembered. Crohn's disease affects the psyche, sexuality, and socialization of afflicted patients. Accustomed to discussions of bowel complaints or rectal bleeding, patient or doctor may never address the serious psycho-social disabilities at the core of chronic disorders. Clinicians must listen to patients with a respectful, non-judgmental attitude. This approach helps validate complaints and assists patients in developing independence and control over disease.

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Hysterectomy in Rhode Island

Anila Verma, MBBS, PhD, Daniel Etheridge, BS, and Jeffrey F. Peipert, MD, MPH

Hysterectomy is a common, yet increasingly controversial surgical procedure for nonmalignant gynecologic conditions in women. In the United States, approximately 600,000 hysterectomies are performed each year with an estimated annual cost of $5 billion.[1] While the total number of hysterectomies in U.S. has fallen since reaching a national high, in 1975, of 724,000, the numbers may rise as the baby-boom generation ages.[2]

Rates of hysterectomy vary widely by geographic region and women's age, race and socioeconomic background. For 1988-1993, national hysterectomy rates were lowest for women in the Northeast (average annual rate of 3.9 hysterectomies per 1,000 women) and highest for women in the South (6.8 per 1,000). During 1980-1993, the average age at the time of hysterectomy was 47.7 years in the Northeast; 44.5 years, the Midwest; 44.0 years, the West; and 41.6 years, the South.[3-5] The American College of Obstetricians and Gynecologists (ACOG) task force attributed these disparities partly to regional differences in gynecologists' training.

This manuscript will provide an overview of hysterectomy rates and approaches to hysterectomy in the United States and at the Women & Infants Hospital of Rhode Island (WIH).

HYSTERECTOMIES IN THE UNITED STATES (1975-1992)

During 1980-1993, an estimated 8.6 million women over the age of 15 years had a hysterectomy. According to the National Center for Health Statistics, the hysterectomy rate was 7.1 per 1,000 women during 1980-1987; by 1987, this rate had decreased to 6.6. Only simple hysterectomy (International Classification of Diseases, Ninth Revision, Clinical Modification [ICD-9-CM] procedure codes of 68.3, 68.4, or 68.5) was evaluated in this report.

From 1988 through 1993, the average annual rate remained stable at 5.5 hysterectomies per 1,000 women. For every year during 1980-1993, the rate was highest for women aged 40-44 years and lowest for women aged 15-24 years. During 1988-1993, the annual rate of hysterectomy for all women aged 40-44 years was 12.9 per 1,000 women.[5-6] In this period, 55% of all hysterectomies were performed on women aged 35-49 years.

Abbreviations Used:

ACOG	American College of Obstetricians and Gynecologists
DUB	dysfunctional uterine bleeding
ICD-9-CM	International Classification of Diseases, Ninth Revision, Clinical Modification
LAVH	laparoscopic assisted vaginal hysterectomies
WIH	Women & Infants Hospital of Rhode Island

The three diagnoses most often associated with hysterectomy were uterine leiomyoma (fibroid), endometriosis and uterine prolapse. Uterine leiomyoma accounted for 61% of hysterectomies among black, 29% among white and 45% among women of other races. Endometriosis and uterine prolapse were listed more often for white women than for black and other races. Interestingly, the hysterectomy rate for uterine leiomyoma among black women was more than twice that among white women (62% vs. 29%), whereas endometriosis and uterine prolapse were more fre-

Table 1. Population Estimates for Rhode Island and the Number & Annual Rate of Hysterectomy at Women & Infants Hospital

Year	Population Estimates*	Total Number of Hysterectomies	Annual Hysterectomy Rate†
1993	284,918	830	2.9
1994	281,509	745	2.6
1995	278,825	773	2.8
1996	278,763	870	2.1
1997	278,351	899	3.2

* Population estimates for female, residents aged over 18 years were obtained from the summary report on population estimates–Rhode Island; and

† Annual rate per 1,000 female, residents aged over 18 years.

Table 2. Comparison between the United States and Women & Infants Hospital for Frequency of Indication for Hysterectomy

Indications for Hysterectomy	United States (%)	W&I Hospital (%)
Malignancy	10	10
Uterine fibroid/leiomyoma	32	40
Endometriosis	6-10	9
Uterine prolapse	10-15	19
DUB	21	7
Pelvic pain	12	5
Endometrial hyperplasia & Cervical dysplasia	6-10	3
Pelvic mass	(Not Reported)	7

quently associated with hysterectomy among white women than among black women.[5] The annual rate of hysterectomy associated with endometriosis and uterine prolapse was highest for white women aged 35-44 years (3.5 and 1.8 per 1000 women, respectively). However, among black women, the rate associated with uterine leiomyoma was 12.1 per 1000 women at 35-44 years - twice the combined rate of both endometriosis and uterine prolapse in white women.

During 1988-1993, the proportion of hysterectomies performed vaginally increased significantly. This route was used for 28% of hysterectomies performed on white women, 15% on black women, and 23% on women of other races. Since leiomyoma are believed to be more prevalent in black women, it is not surprising that these larger uteri are more often removed via the abdominal route. In 1990s, 32% of the hysterectomies in white women were performed by the vaginal route compared with 26% during the 1980s. The trend in early 1990s in the increased usage of vaginal hysterectomy overlaps with the rise in simultaneous ICD-9 coding for laparoscopy and vaginal hysterectomy. In 1992-1993, approximately 15% of the vaginal hysterectomies were associated with laparoscopy. Later studies have found the rate of laparoscopic assisted vaginal hysterectomies (LAVH) as high as 30-40% of the vaginal hysterectomies.[7]

HYSTERECTOMIES AT WOMEN & INFANTS HOSPITAL

The George Anderson Outcome Measurement Unit collected data for hysterectomies at WIH from the Hospital Discharge Database, Hospital Association of RI and National Perinatal Information Center. To compute hysterectomy rates at WIH, all women over the age of 18 years in Rhode Island were included in the denominator. Since all women at and over 15 years are included in the denominator for United States annual hysterectomy rates, the national rates will be lower than the WIH rates.

To determine the frequency of indications for nonmalignant conditions for hysterectomy, the following seven diagnoses were identified from the hospital database using ICD-9 disease codes: uterine leiomyoma (fibroid), endometriosis, uterine prolapse, dysfunctional uterine bleeding (DUB), pelvic pain, pelvic mass, and endometrial hyperplasia plus cervical dysplasia.

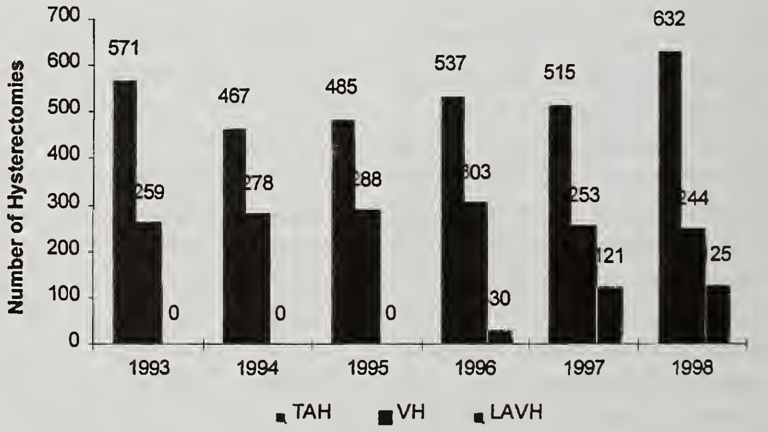
The four race categories from RI used to analyze data were: white, black, Hispanic and others including Asian, American Indian, Alaskan and unknown. (In the "other" category, the largest group was the "unknown.") Race was self-reported at the time of hospital admission. Women's age was categorized as <35, 35-44, 45-54 and >54 years. Procedures were categorized by approach to hysterectomy: abdominal, vaginal, and LAVH.

RESULTS

Between October 1996 and September 1997, 2014 hysterectomies were performed in Rhode Island hospitals for both malignant and nonmalignant indications - a rate of 7.2 per 1000 women above the age of 18 years; 58% (1168 of 2014) of these procedures were performed at WIH. In earlier years, 48-50% of the total RI hysterectomies were done at WIH. Using the population estimates for Rhode Island from 1993 through 1998, Table 1 shows the steady increase in the absolute number and rate of hysterectomies at WIH from 1994-1998.

Table 2 compares the frequency of indication for hysterectomy at WIH and nationally. Although the percentage of hysterectomies performed for malignant conditions is similar in the two groups (10%), WIH reports a higher percent-

Figure 1. Approach to Hysterectomy at Women & Infants Hospital in 1993-1998



TAH - Total abdominal hysterectomy; VH - Vaginal hysterectomy; and LAVH - Laparoscopically assisted vaginal hysterectomy; separate coding for LAVH started in late 1996.

age of hysterectomies for uterine leiomyoma (fibroid) and prolapse, and a lower percentage for the remaining indications including DUB, pelvic pain and endometrial hyperplasia plus cervical dysplasia.

Table 3 contains the distribution of indication for simple hysterectomy performed for non-malignant indications at WIH, by age categories. In the less than 35 year-old group, the top four indications for hysterectomy are endometriosis, dysfunctional uterine bleeding (DUB), leiomyoma, and pelvic pain. Between 35 and 54 years, the majority of hysterectomies are performed for leiomyoma. Over the age of 54, almost 60% of the hysterectomies were due to pelvic organ prolapse. Statistics from 1997 show that for non-malignant conditions (N=899), the most frequent age group for hysterectomy was 45-54 years (32%), followed by the age group 40-44 years (27%). Average age for hysterectomy at Rhode Island hospitals is 46.5 years, close to the average age of 47.7 years for the Northeast.

Table 3. Frequency of Indication for Hysterectomy by Age Categories

Indication for Hysterectomy	<35 years	35-44 years	45-54 years	>54 years
Leiomyoma	20.2%	54.3%	61.6%	11.4%
DUB	21.7%	12.4%	6.3%	1.3%
Endometriosis	26.1%	11.4%	4.5%	–
Pelvic Pain	14.4%	3.3%	1.4%	2.7%
Pelvic Mass	4.3%	3.8%	5.6%	18.1%
Prolapse	8.7%	10.9%	17.1%	59.1%
Cervical Dysplasia & Endometrial Hyperplasia	1.4%	1.5%	2.4%	6.0%

Surgical approach to hysterectomy was analyzed for WIH using ICD-9 CM-684 (total abdominal hysterectomy), CM-6851 (LAVH), and CM-6859 (vaginal hysterectomy). LAVH and vaginal approach were classified under the same code until the last quarter of 1996. Since 1994, there is a trend toward the increased use of either the vaginal or laparoscopic approach (LAVH) to hysterectomy. (Figure 1)

We stratified the surgical approach by race. Among black women, 69% of hysterectomies were performed using the abdominal approach. In white and Hispanic women undergoing hysterectomy, 58% and 52% were performed abdominally. This difference is probably due to large myomatous uteri in black women. Similar to national figures, 66% of the vaginal hysterectomies were performed for the indication of uterine prolapse and 18% for fibroid uterus; whereas 61% of the abdominal hysterectomies were done for fibroid uterus and 1.3% for prolapse.

The patient's average age, hospital charges and length of stay are shown by surgical approach to hysterectomy in Table 4. The hospital charges include fees for room and boarding, surgical services, medical supplies, and ancillary services like laboratory tests. Fees for professional services are not included.

Table 4. Average Age, Hospital Charges and Length of Stay

Average	Abdominal	Vaginal	LAVH*	p-value
Age (years)	44.6	51.6	43.7	0.006[†]
Hospital Charges ($)	7595	6138	6381	0.002[‡]
Length of Stay (days)	3.7	2.5	1.9	0.0001[Ω]

* Laparoscopically Assisted Vaginal Hysterectomy;
[†] p-value for difference between vaginal verses LAVH group;
[‡] p-value for difference between abdominal versus vaginal hysterectomy group; and
[Ω] p-value for difference between abdominal versus vaginal hysterectomy, and abdominal versus LAVH group.

DISCUSSION

The optimal use of hysterectomy is a topic of discussion within the medical profession. Various studies support the conclusion that the nature of society and health-care providers, more than pathology, dictate the rate and route of surgical procedures.[8,9] Variation in hysterectomy rates may be attributable to psychosocial factors that influence demand and supply. Provider factors are important too. Professional uncertainty has been cited as a key explanation for wide variations in rates of common surgical procedures.[10] Uncertainty regarding hysterectomy is also attributable to the present lack of data on decision-making for hysterectomy and those on patient-centered outcomes following hysterectomy.

In the past, approximately 50% of the total hysterectomies in RI were performed at WIH. However, in 1997 statistics, 58% of the total hysterectomies in RI were performed at WIH. The number of gynecologists with privileges at WIH increased from 118 in 1993 to 131 in 1997. We believe that the increase in rate of hysterectomy at WIH can be attributed to the increase in the number of surgeries referred to WIH from other facilities and not to escalation in the rate of hysterectomies. The annual rate of 7.2 per 1000 women in RI is higher than the national rate of 5.5; but the actual disparity in rates may be much less, considering the larger denominator for estimating the annual rate in RI.

Results (Table 2) reveal that the proportion of women undergoing hysterectomy at WIH for leiomyoma is higher than the national statistics, whereas that due to DUB, pelvic pain and endometrial hyperplasia plus cervical dysplasia is lower. The average age for hysterectomy is higher in Rhode Island than the average age in the Midwest, the West and the South. This corresponds with the results that at WIH uterine leiomyoma is the most common indication for hysterectomy in the age group 45-54 years, unlike the other geographical regions, where the most common age for this indication is 40-44 years.

Several studies have reported

younger average age for hysterectomy, and higher rate of abdominal than vaginal approach to hysterectomy among black women compared to other races.[11,12] Despite limited number of black subjects in our population (4% of the total hysterectomies), our findings support both conclusions. The higher prevalence of leiomyoma in black women may explain these findings. Large myomatous uteri are more often approached via the abdominal route.

Similar to the national trend, hysterectomy by vaginal approach has become more popular at WIH. Further, women having LAVH were younger than those having hysterectomy by vaginal or abdominal approach, had much shorter length of stay, and higher hospital charges compared to vaginal hysterectomy. However, abdominal hysterectomy was found to be the most expensive procedure, due to longer length of stay. A detailed study of quality of life, health care utilization and costs among women undergoing hysterectomy in a managed care setting also found highest costs during hospitalization among women undergoing abdominal hysterectomy.[13] Several studies[14,15], though, have concluded that the costs that include hospital charges, professional fees, operative time, and instrumentation are highest in the LAVH group.

Previous researchers including Kjerulff KH et al[11-12] and Carlson KJ[16] have adequately described hysterectomy and associated outcomes. Numerous Rhode Island researchers are addressing the issue of patient-oriented outcomes after hysterectomy and alternatives to hysterectomy. Dr. Kay Dickersin, Associate Professor of Community Health at Brown University, is leading an ongoing investigation funded by the Agency for Health Care Policy and Research to evaluate hysterectomy versus endometrial ablation for dysfunctional uterine bleeding.

What remains to be investigated is the relationship of various approaches to hysterectomy with patient-centered outcomes. Investigators at the George Anderson Outcome Measurement Unit at WIH are developing projects on key issues; specifically, a study of focus groups of physicians and patients on preferences and decision making for hysterectomy (funded by the Agency for Health Care Policy and Research); a retrospective study of febrile morbidity following hysterectomy; a survey of physicians' views regarding hysterectomy and evidence-based practice guidelines (to include all Rhode Island gynecologist-members of ACOG); and a longitudinal study of the impact of surgical route to hysterectomy on woman's quality of life.

Some of the problems associated with geographical and racial differences in hysterectomy rates may be addressed by developing uniform clinical practice guidelines. However, there is no known acceptable hysterectomy rate in the population. What is most important is how the procedure will affect a woman's quality of life. Physicians may assist by not only offering appropriate alternatives to surgery, but also by promoting the optimal surgical approach(es) to hysterectomy.

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Rhode Island
Quality Partners, Inc.

The PRO Program in the New Millennium

Barbara Miller Perry, RPh, MPH

In 1992, the Health Care Financing Administration's (HCFA) Peer Review Organization (PRO) Program introduced the innovative Health Care Quality Improvement Program (HCQIP) to work collaboratively with physicians, hospitals, and other parties to enhance quality of care and health outcomes for Medicare beneficiaries. Rhode Island Quality Partners (RIQP) has been the Medicare contractor implementing this concept in our state since 1996.

As the Peer Review Organization/Quality Improvement Organization (PRO/QIO) for Rhode Island, RIQP provides objective, continuous quality evaluation and improvement programs through partnerships with hospitals, managed care organizations, physicians, and long-term care facilities. We have also established external partnerships with the medical society, state agencies, advocacy groups, professional and volunteer organizations, and universities to further the mission of improving quality of care and outcomes for Medicare beneficiaries. As we have described in previous articles, this effort has resulted in improving the quality of care provided to patients hospitalized with acute myocardial infarction (AMI) or pneumonia; increased use of preventive services like influenza immunization; and mammography screening for breast cancer.

Abbreviations Used:

AMI	acute myocardial infarction
CASPROs	Clinical Area Support PROs
DQIP	Diabetes Quality Improvement Project
HCFA	Health Care Financing Administration
HCQIP	Health Care Quality Improvement Program
PEPP	Payment Error Prevention Program
PRO	Peer Review Organization
PRO/QIO	Peer Review Organization/Quality Improvement Organization
QISMC	Quality Improvement System for Managed Care
RIQP	Rhode Island Quality Partners

Table 1. National Health Improvement Clinical Topics and Quality Indicators

Clinical Topic	Quality Indicators
Acute Myocardial Infarction	Aspirin within 24 hours of admission Aspirin at discharge Beta blockers within 24 hours of admission Beta blockers at discharge ACE inhibitors for low LVEF Time to initial reperfusion Documentation of smoking cessation counseling provided during hospitalization
Heart Failure	Appropriate assessment and treatment for low LVEF
Pneumonia	Time to initial antibiotic administration Appropriate administration of antibiotics for 'atypical' pathogens Influenza immunization Pneumococcal (PPV) immunization
Stroke/Transient Ischemic Attack/ Atrial Fibrillation	Aspirin/antiplatelets for stroke/TIA Warfarin for atrial fibrillation Reduce inappropriate use of rapid acting antihypertensive agents
Diabetes	Periodic retinal exam by an eye professional Annual HbA1c testing Appropriate assessment of nephropathy Biennial testing of lipid profile
Breast Cancer	Biennial screening mammography

In the new millennium, HCFA will be implementing a definitive national effort to improve the quality of care for Medicare beneficiaries in six clinical areas (Table 1). This is the only organized effort of this magnitude and intensity to be launched in this country. The PRO in each state will play a key role in this effort under what is called the 6th Scope of Work. This will begin in Rhode Island in the late fall of 1999. The national clinical topics and the quality indicators to be addressed in each of them were selected based on their clinical strength, high prevalence, strong evidence, and measurability and the major impact on the Medicare population. HCFA will provide statewide baseline performance rates for each quality indicator to the PROs by the start of the new contract period. We look forward to working directly with Rhode Island physicians over the next three years to improve those rates where the opportunity to do so exists. As you can see, three of the national clinical topic areas touch upon care of seniors in the outpatient setting - e.g. pneumonia and influenza immunization, diabetes and breast cancer.

With this new effort, the opportunity will now exist for national comparison of clinical performance measures and opens exciting new possibilities in the exchange of information and ideas. RIQP has participated in several multistate and one national project under the 5th Scope of Work and experienced firsthand the benefits of collaboration at this level.

To lead the national efforts of the PRO program, HCFA has created six Clinical Area Teams to coordinate the quality improvement efforts for each national topic. The teams will serve as national content experts. In addition, six PROs, one for each clinical topic, will provide local PROs the opportunity for input into the effort. These PROs are called Clinical Area Support PROs (CASPROs). Qualidigm, our sister organization in Connecticut, is the CASPRO for AMI.

In addition to national projects, RIQP will initiate local quality improvement projects in response to interests, needs and opportunities within the state of Rhode Island. The setting for local projects will be outside the traditional acute-care hospitals and Medicare+Choice plans, and may include alternate settings like long-term care facilities and home health agencies. Attention will also focus on reducing the disparity in the quality of care that exist between disadvantaged groups and all other beneficiaries living in Rhode Island.

As part of Medicare's Quality Improvement System for Managed Care (QISMC), the Medicare+Choice plans will also be implementing quality improvement projects. In 1999, the plans will conduct two quality improvement projects. One project will address the care of diabetic patients using the quality indicators established by the Diabetes Quality Improvement Project (DQIP) developed by the PRO Program. This offers RIQP the opportunity to continue to work with the plans in the area of diabetes and other clinical focus areas such as prevention and care of acute and chronic conditions, high-volume and high-risk services, and continuity and coordination of care.

The analyses upon which this publication is based were performed under Contract Number 500-96-P519, entitled "Utilization and Quality Control Peer Review Organization for the State of Rhode Island," sponsored by the Health Care Financing Administration, Department of Health and Human Services. The content of this publication does not necessarily reflect the views or policies of the Department of Health and Human Services, nor does mention of trade names, commercial products, or organizations imply endorsement by the U.S. Government.

The author assumes full responsibility for the accuracy and completeness of the ideas presented. This article is a direct result of the Health Care Quality Improvement Program initiated by the Health Care Financing Administration, which has encouraged identification of quality improvement projects derived from analysis of patterns of care, and therefore required no special funding on the part of this Contractor. Ideas and contributions to the author concerning experience in engaging with issues presented are welcomed.

RIQP's strict adherence to confidentiality and disclosure requirements under the 5th Scope of Work will continue to be a top priority for RIQP. This commitment gives our collaborators the safety and security to identify areas of improvement and develop successful intervention strategies.

A new addition to the 6th Scope of Work that has generated considerable controversy is the Payment Error Prevention Program (PEPP). As a means to protect the integrity of the Medicare Trust Fund, the PROs will develop programs, primarily educational, to prevent payment errors. RIQP is committed to working with our collaborators in the same spirit of continuous quality improvement in this area. As of this writing, it appears that this aspect of the PRO program will relate to facilities rather than individual physicians.

This overview of the 6th Scope of Work highlights the new opportunities ahead for RIQP and our collaborators and partners. It is gratifying to know that our current successes have laid the foundation for continuing to advance the quality of care in Rhode Island. We also welcome the participation of new partners in our mission to enhance the health and well-being of Medicare beneficiaries and residents of Rhode Island.

For more information on the 6th Scope of Work, visit the HCFA website at www.hcfa.gov/quality/qlty-5b4.htm#sow.

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Health by Numbers

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Jay S. Buechner, PhD, and William J. Waters, Jr., PhD

Utilization of Preventive Dental Services

Jay S. Buechner, PhD, Hanna Kim, PhD, and Jana E. Hesser, PhD

Regular visits to a dentist for prevention and screening are a key component of good dental care. Such visits can commence as early as age two and should continue throughout life. Increasing third-party coverage of dental care costs, usually provided as a benefit of employment or through enrollment in the Medicaid program, has reduced the economic barriers to routine preventive dental care. Nevertheless, substantial proportions of persons of all ages do not receive annual preventive care. In this analysis, the utilization rates for preventive dental care for Rhode Island during 1996, with selected comparisons to 1990, are presented as a function of age, income, and presence of third-party dental coverage.

Methods

In both 1990 and 1996, the Rhode Island Health Interview Survey was performed by telephone and obtained information on all members of participating households, including demographic, social, and economic characteristics, health status, health-related practices, health care utilization, and coverage for health care costs. In 1990, 6,536 persons were included in the survey; in 1996, 6,583 were included. For each person ages two years and older, it was determined when he or she had last been to a dentist and whether any visit during the previous 12 months had been for preventive care. It was also determined whether he or she was covered for the costs of dental care by any dental insurance plan or other type of health plan, including government programs. All data were either self-reported or reported by a knowledgeable member of the household and have not been otherwise validated.

Results

In 1996, 72% of Rhode Islanders ages two and older had been to a dentist during the previous 12 months for preventive care, up slightly from 70% in 1990. By age, this proportion varied between 48% for those ages 75 and older and 90% for those ages 6 through 17 years. (Figure 1) Among adults, the percentage was highest among those ages 35 through 44 years and relatively low among the elderly.

The likelihood of receiving preventive dental care depended strongly on household income. (Figure 2) Persons in households with incomes below the federal poverty level were approximately three times as likely to lack such care as persons in households at the highest income levels. At each intermediate income level, utilization rates increased with increasing income.

In 1996, 61% of those in the survey were reported as having third-party coverage for the costs of dental care, up from 53% in 1990. For those covered by a commercial dental plan or other non-governmental plan, the utilization of preventive dental services was higher than for others. Utilization by those covered by the Medicaid program was similar to utilization by those with no coverage. (Figure 3) Between 1990 and 1996, the percentages in each group receiving preventive care changed only slightly except

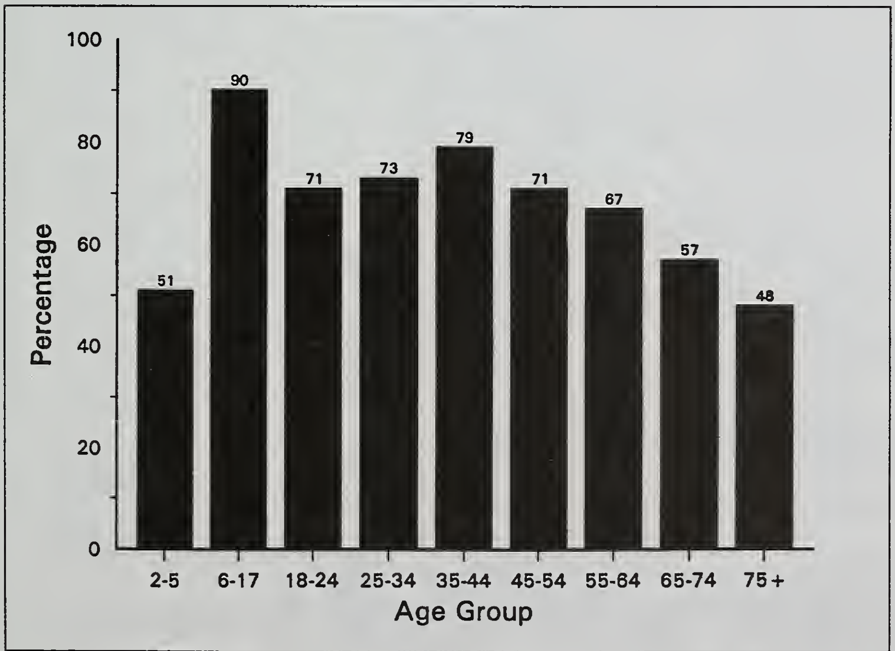

Figure 1. Percent of Persons with a Preventive Dental Visit within the Previous 12 Months, by Age Group, Rhode Island, 1996.

for those with Medicaid, for which there was a small, but significant, increase.

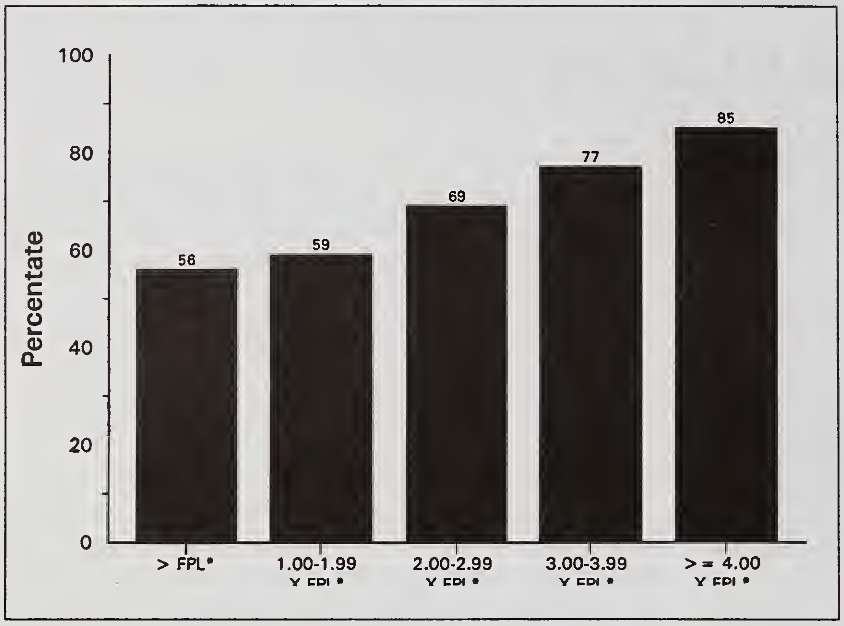

Figure 2. Percent of Persons with a Preventive Dental Visit within the Previous 12 Months, by Household Income Range, Rhode Island, 1996.

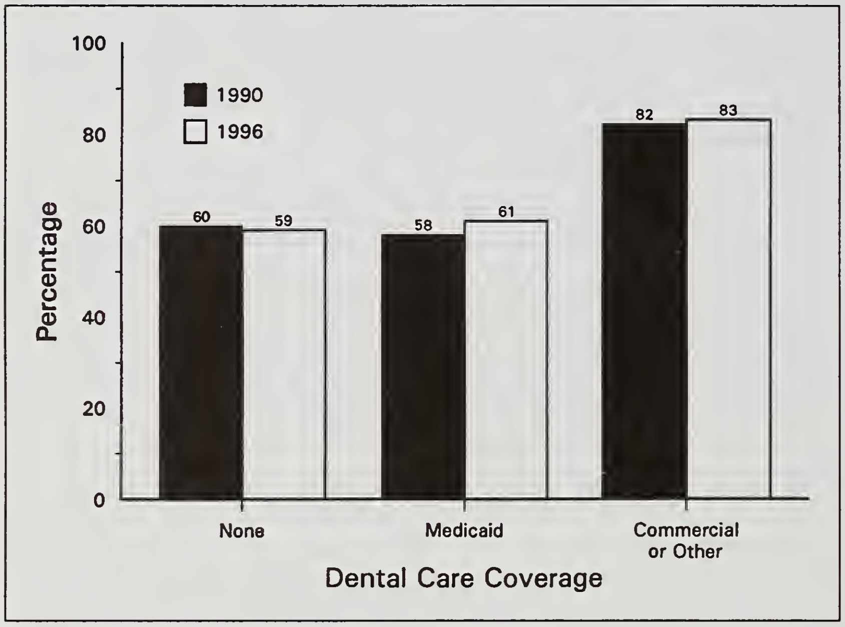

Figure 3. Percent of Persons with a Preventive Dental Visit within the Previous 12 Months, by Dental Care Coverage, Rhode Island, 1990 and 1996.

Discussion

Over one-quarter of all Rhode Island residents are reported as not receiving preventive dental care within the recommended interval of twelve months. For some, notably those with low incomes and those without third-party coverage for dental care costs, it can be assumed that the barriers to such care are, at least in part, financial. For others who have not received preventive care despite having coverage and/or .higher incomes, the barriers may involve other issues of access, such as the availability of dentists who participate in their plan, as well as issues of awareness and motivation. Further information on the specific barriers to such care are needed in order to help determine how to increase the percentage of Rhode Islanders who receive such care within the recommended time period.

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PUBLIC HEALTH BRIEFING

Rhode Island Department of Health
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Edited by John P. Fulton, PhD

Resistance Exercises in Older Adults

Carol Ewing Garber, PhD

Muscular strength declines with age, although there is variability in the rate of decline in various muscle groups. In both men and women, there is a decrement in muscle strength of approximately 30% between the ages of 20 and 70 years, with much of the loss occurring after age 45-50 years and during menopause, although estrogen replacement therapy may attenuate this loss in women.[1,2] There are few data on strength in people older than 70 years.[1,2]

BENEFITS

Many studies have demonstrated that resistance training will significantly increase muscular hypertrophy and strength in healthy and frail old participants.[1,2,3] The relative magnitude of the increases in the elderly appears to be similar to younger subjects, when differences in initial strength are considered.[2,3] In older adults, these increases in strength often translate into improvements in the ability to ambulate and to carry on the activities of daily living.[3,4] Thus, older individuals can benefit greatly from participation in a regular program of resistance training exercise.[2,3,5] The American College of Sports Medicine (ACSM) recommends strength training for all healthy adults;[2,3] the American Heart Association recommends resistance training for patients with stable cardiovascular disease.[6]

TYPES OF RESISTANCE TRAINING

Resistance training exercise generally involves lifting weights, all or part of one's body weight, or moving the body against some externally imposed resistance (e.g., elastic re-

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sistance bands or a Nautilus-like machine). Resistance training involves two types of muscular contraction: isometric and isotonic. Isometric, or static, exercise involves muscular contraction without shortening of the muscle or movement of the limb, and usually occurs at the beginning and at the end of the range of exercise. Isotonic, or dynamic, exercise involves muscular contraction accompanied by movement of the limb. The isotonic contraction can be concentric, where the muscle shortens, or eccentric, where the muscle length lengthens, usually when moving against gravity or against heavy resistance, i.e. walking down stairs, or lowering a heavy weight. Eccentric contractions generally result in greater muscle soreness and should be minimized, particularly in the earlier phases of an exercise training program.

Current ACSM recommendations for resistance training suggest exercising at a "sufficient intensity to improve muscular strength and endurance and to maintain fat-free mass."[2] Exercises for all the major muscle groups should be included and the exercise program should be individualized and progress gradually. One set of 10-15 repetitions of each exercise two to three days per week is recommended for adults over age fifty. The amount of resistance can be determined by the amount of weight or resistance that can be lifted comfortably at least 10 times.

STRETCHING

Stretching exercises are strongly recommended before and after performing the resistance exercises. It is helpful to warm the muscles by performing some light aerobic or calisthenic activity before engaging in the exercises. Many patients will benefit from individualized instruction by an exercise physiologist. YMCAs, health clubs, and other community facilities offer these services. For home exercise, common household objects can be used: cans of various sizes, milk jugs with varying amounts of water added, bicycle tubes, or elastic bungee cords (without metal hooks) can all become training tools. Hand, wrist and ankle weights of varying weight can be purchased inexpensively at many department stores. Older adults often do better with attachable wrist and ankle weights that allow for adjustment.

HOW MUCH WEIGHT?

For each exercise, the patient should choose a weight that they can lift comfortably 10 times. (The amount of weight that an individual can lift may vary with different muscle groups.) The exercises should feel "somewhat hard" to "hard," and the patient should not strain to complete the exercises. To improve exercise adherence, new exercisers generally should begin with a less intense program (lower resistance and repetitions). A gradual progression, with increases in the amount of weight or resistance every 2-4 weeks, is usually well tolerated. A good gauge for progression is when the patient can lift the weight or repeat an exercise 20 times, and the level of intensity feels "fairly easy."

MUSCLE SORENESS

Patients may experience some muscle soreness 24-48 hours following exercise at the beginning and when increasing the training, but this should not be severe or incapacitating. If they do experience severe soreness, they should probably reduce the amount of resistance and/or repetitions. Mild fatigue is not uncommon in the early stages of exercise, but the patient should not experience inordinate fatigue, which may signify over-training at an intensity or duration of exercise that is too high, or it may signify exercise intolerance in some patients, such as those with heart failure.

GETTING STARTED

An exercise program should begin at a level consistent with the patient's habitual physical activity. That is, an individual who does little physical activity in the course of his/her day generally needs to begin with low intensity exercise with fewer repetitions, while someone who is active throughout the day, including lifting and walking or moving about, can begin at a higher intensity. Totally inactive individuals can start with calisthenic exercises or perform the motions without any weight or resistance for a week or two. Then light weights or resistance can be added gradually.

Prior to recommending exercise training, contraindications to exercise,[6] co-morbidities, and/or problems with balance, vision, and cognition must be considered. Patients with balance problems can exercise in a sitting position. Patients with visual impairments will need instruction in audio or large print format. Patients with cognitive deficiencies need close supervision and frequent repetition of instructions. Older adults with coronary artery disease, COPD, or diabetes mellitus may benefit from medically supervised programs.

Physicians can encourage older patients to begin a comprehensive exercise program, including resistance training. Many older patients, with few exceptions, can safely engage in resistance training and can realize substantial improvements in strength, balance, and the ability to carry out activities of daily living.

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Medicine ABROAD

From the Ground Up: The Evolution of a Hospital in the Third World

Mary J. Bliss

With 160,000 people, La Romana, a tourist-oriented city in the Dominican Republic, boasts the largest sugar cane company in the world. However, a poverty-stricken population of sugar cane workers receives little or no health care.

Some key people in the local community and multiple volunteer American medical and construction workers have together spearheaded the creation of the Good Samaritan General Hospital in La Romana.

Reverend Jean Luc Phanord, a leader of the local Haitian community, was the hospital's founder: he recognized that the most extremely impoverished, who are primarily Haitian immigrants, could not afford services at the other hospitals in La Romana; hence, they were not receiving treatment.

Reverend Phanord initially raised money to buy an eight acre plot of undeveloped land in a run-down area adjacent to the city. The building of the hospital began in 1989. It has been funded and constructed entirely by American volunteers, and with every visiting American construction team the hospital has grown. Institutions such as Rhode Island Hospital and the Lahey and Mayo Clinics have donated and shipped all equipment and supplies. At this point the first floor of the hospital has been completed. The ambulatory clinic officially opened on November 9th, 1997.

THE BATEYS

"Bateys" are the small villages where the sugar cane workers and their families live on a subsistence level. Men are recruited from Haiti to come work in the cane fields, since the cost of manual labor is less than that of machinery. (About 90% of the twenty-five thousand people living in the bateys are Haitian.) The immigrants, almost all of whom are illegal, arrive with their families and sign on to a life of brutally hard labor. They live in tiny, filthy, makeshift shacks without electricity or running water. The communal latrines are shallow trenches, often located next to the primary water supply. Most of the inhabitants do not have access to medical care. There are over one hundred bateys in the La Romana area alone, each one having between 500 - 1500 residents.

Abbreviations Used:

GSGH Good Samaritan General Hospital

The GSGH was created with this population in mind.

One service provided by GSGH is a mobile clinic that travels to these rural areas several times per week. A fundamental source of the health care comes from volunteer American medical workers. Medical teams from RI have been involved with the project since 1992. A team of 40-50 medical and construction volunteers from RI travels annually to La Romana and works for up to two weeks.

My first trip to the bateys was with Reverend Phanord's wife Elza, a public health worker, in 1996. We drove for about an hour and a half along bumpy dirt roads in an old family jeep early one morning to visit two of the villages. Outside of the city are endless rolling fields of blue-green sugar cane that shimmer against the expansive, deep blue sky. We passed many weathered-looking people along the way, with most of the women balancing impossibly huge packages on their heads. We also passed several garbage dumps where starving horses and pigs foraged for food.

The first batey we stopped at was called "Lima." As we drove up we heard a swelling chant of "Americana, Americana!" which referred to me in the back seat. When I stepped out of the jeep I was instantly surrounded by children. They were pointing at me and laughing and all wanted to touch my white skin, which would make them shriek and laugh even harder. A few reached up to touch my red hair, which made them squeal with disbelief. Any attempt on my part to speak Spanish was relentlessly mocked and then mimicked in a silly voice. Every child wanted to hold my hand, so when I walked anywhere I had a different child attached to all ten fingers! Some of the children surprisingly had blondish-reddish hair, similar to the Aborigines in Australia. (This is a sign of severe anemia.) Most of the children were barefoot and wore only the remnants of donated American clothes.

The GSGH oversees the monitoring of the growth rate of the babies and toddlers to determine who is malnourished. Each month the babies get weighed. We placed them in a little sack and put them up on a scale on the wall. None of the toddlers weighed over 17 Kg. Most weighed under 10. Many had distended bellies signifying nutritional deficiencies.

"Batey 203" seemed even worse off than Lima. One half-naked little girl was sitting on a tiny chair in the middle of a footpath, holding a loudly crying baby. The girl could not have been more than three or four herself. An emaciated woman emerged from a tiny hut and brusquely pulled the baby out of the girl's thin arms and brought the still crying infant over to us. I held her for a few minutes and by what must have been pure coincidence she stopped crying. She felt as though all she wanted was some warmth and security. Her little nose was running and I detected a rasp in her tiny chest when I patted her back. The delicate little creature turned out to be six months old - I have seen bigger two month-old infants.

Various medical teams from the U.S. volunteer their services to the GSGH and the bateys throughout the year. When the medical teams visit, an average of 150 - 200 people are seen each day. A large batey will have approximately 350 children (age 0-18) while a smaller one will have on average 170. The most common complaint for children and adults is stomach pain, which is generally due to parasites, gastritis, or simply hunger. Many of the women, especially of child-bearing age, are severely anemic, with hemoglobins of 6-10 mmHg. Diabetes is also common, with subsequent visual deficits. Most of the children are underweight and lethargic, often due to not consuming enough calories.

Demographic Breakdown:

Atlas de la Salud, Republica Dominicana 1995

1993	**Dominican Republic**	**La Romana**
Total Population	7089039	157964
Urban	3935545	136677
Rural	3153493*	21287*

La Romana Heath Care Facilities:

Total Establishments	15
Hospitals	2
Clinics	1
Consults	7
Rural Clinics	5

* numbers rough

THE HOSPITAL

In January, 1998, the GSGH hired one local Haitian doctor to work in the clinic full time, six days a week. On average 25 - 30 people are treated each day. Like the population in the bateys, the most common complaints are of non-specific gastritis, anemia, and hypertension. The most generic consumables received by all patients treated are anti-inflammatories, antihypertensives, parasite medicine and vitamins.

In addition to the resident physician, the current staff includes the Hospital Administrator, Chief Accountant, two secretaries, and one pharmacy worker. Local doctors and nurses are commissioned separately to work in the bateys. The projected salary for clinic physicians at the hospital is approximately $4500 annually. The goal is for the hospital to pay these salaries and those of the doctors hired to work in the bateys with money earned in the ambulatory clinic. The cost of a general consult at the hospital is $3.20 (50 pesos) while in the bateys patients are charged between $0.19 - $1 (3 - 15 pesos), depending on the relative poverty status of the batey.

Presently the primary source of income is derived from medications donated from the U.S: the hospital sells them at a maximum of 1/3 of the current rate found at any of the local pharmacies. For example, if a 50 pill bottle of aspirin is donated, 30 pills are marked for the bateys while the remaining 20 are sold at the hospital pharmacy. This system has been able to almost meet the costs of running the clinic, although the GSGH continues to rely on American donations.

1999 BUDGET

In 1999 it is projected that between the visiting American groups and the local doctors at the hospital, 2635 patients will be seen in the hospital clinic and 38,751 in the bateys. An additional 234 patients will also be treated by American doctors outside of the hospital for surgical procedures at a separate clinic. This gives a total of 41,620 patients treated in connection with GSGH.

The projected annual budget for 1999 totals $80,140 (1,242,176 pesos). The projected income totals $93,048. However, this figure includes an expected $22,279 in U.S. donations in addition to the volunteer medical help and donated supplies. Without these extra funds the hospital would only expect to gross $70,769 - $9371 short of the projected income.

FUTURE PLANS

In 1999 GSGH plans to open its laboratory, which is close to being fully equipped. Within the next two years the hospital hopes to add two surgical suites. There also are plans to add a pediatrician, a cardiologist and a gynecologist as well as a part time (weekly) ophthalmologist and dentist. Eventually the hospital hopes to grow into a full care facility with at least three additional floors for inpatient stays.

CONCLUSION

The sugar cane workers and the indigent of La Romana live under appalling conditions. The GSGH is the first hospital in the Dominican Republic designed with the primary goal of providing health care to the people in the bateys. It is unusual because it does not rely on government funding but is supported solely from American donations. Right now the hospital is in a critical growing stage. It wants to develop into a full care facility. Once the hospital is fully operational, it hopes to be financially independent, no longer tied to United States aid.

The public hospitals in the Dominican Republic are generally of poor quality, medically and administratively. Patients are expected to provide their own bedding, food, and often their own medicine. Hospital management information in developing countries is also difficult to obtain due to a lack of standardized information systems. Results from a study on public hospitals in the Dominican Republic showed all surveyed to be grossly inefficient, chaotic, and poorly managed.[1] The authors found the budget of the Aybar Hospital in Santa Domingo to be over 50% higher than the actual service costs, demonstrating terrible waste and inefficiency. The GSGH is a small, modest institution, but as it grows it will have to eliminate some of the inefficiency common to other health institutions in the Dominican Republic.

The GSGH is an example of a community that has taken the initiative to provide its own non-governmental system of health care, to help a population that needs it the most. If the GSGH is a success, the knowledge gained from the growth process can be shared.

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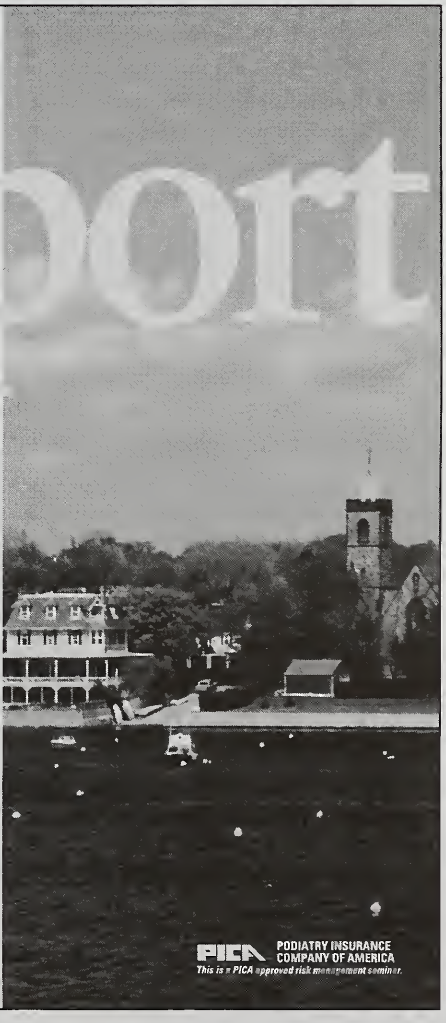
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Judicial Diagnosis

Antitrust and Physicians

Jeffrey Wishik, MD, JD

Several solo orthopedists (or neurosurgeons or obstetricians....) meet to discuss the latest reimbursement cuts by Huge Healthcare Company. Soon thereafter, each physician resigns from the provider network. They decide to form a group practice, which will comprise almost all orthopedists in the region. Do these actions have anything to do with antitrust law? The answer is yes. As physicians struggle with the recent changes in the health care industry, they increasingly risk running afoul of antitrust law. Faced with the need to compete more effectively, physicians must understand the limits of acceptable economic behavior.

Many years ago, physicians were self-regulated. Strong ethical codes controlled physician conduct, and there was little governmental involvement beyond licensing laws. Antitrust laws were not applied to physicians because as members of a "learned profession" they were not considered in trade or commerce. The United States Supreme Court eliminated this exemption in 1975.[1] Individual physicians are regarded as competitors, and they are subject to antitrust scrutiny. Price-setting agreements and concerted refusals to deal with insurers (group boycotts) are among the prohibited acts. Unfortunately, these violations are among the easier ways to increase negotiating power. Physicians must somehow integrate their practices if they want to negotiate jointly. Participants in a properly formed physician joint venture are not competitors and may speak with a combined voice. However, if the group is too large or otherwise interferes with competition, it may violate other aspects of antitrust law.

> *Physician ventures without the necessary financial risk-sharing or clinical integration are not likely to be approved.*

Beginning with the 1890 Sherman Act, the antitrust laws sought primarily to increase competition and consumer choice. Although the patient is the ultimate "consumer" of medical services, health plans and insurers are the economic consumers for most antitrust purposes. Three federal laws cover anticompetitve activity.[2] Section 7 of the Clayton Act[3] prohibits mergers that may substantially reduce competition or tend to create a monopoly. Section 5 of the Federal Trade Commission Act[4] outlaws all "unfair methods of competition." Finally, section 1 of the Sherman Act[5] bars all contracts, combinations or conspiracies in restraint of trade while section 2[6] prohibits monopolies and attempts or conspiracies to monopolize. Violations can lead to civil and criminal charges, with treble monetary damages.

Abbreviations Used:

DOJ	Department of Justice
FTC	Federal Trade Commission

Solo practitioners are not regarded very differently than large corporations when it comes to antitrust issues. The core requirement is that individual economic entities - including physicians - must exercise independent judgment. Two or more independent physicians cannot make economic agreements that restrain trade. The practice of medicine may be part art and part science; it is also "trade." Thus, if several physicians leave a health plan after a small policy change is announced by an insurer and discussed later at a physicians' meeting, concerted action may be inferred. There need not be any explicit understanding about the action among the physicians. Indeed, anticompetitive agreements are rarely formal or written. On the other hand, if the policy change were major - a 50% fee reduction, for example - the government might not infer a conspiracy because there is a clear justification for each physician to reach the same decision independently.

There are guidelines to help physicians avoid antitrust violations. Some are from decisions in federal cases, especially United States Supreme Court decisions. However, there is a paucity of cases because these laws have only been applied to health care since 1975. The courts have not clarified issues such as how much integration of practices or financial risk sharing is needed to avoid creating "sham" networks. That task falls to the antitrust divisions of the Department of Justice (DOJ) and Federal Trade Commission (FTC). Physicians can obtain DOJ business review letters or FTC advisory opinions regarding proposed ventures. The agencies indicate whether there will be a challenge to the legality of a proposal. Violations in existing ventures may be corrected through a negotiated consent decree instead of a trial; these are available for review. Finally, explicit guidelines and clues to the agencies' thinking are found in the revised DOJ & FTC Statements of Antitrust Enforcement Policy in Health Care (*Statements*).[7]

Since all business agreements involve some restraint, the Sherman Act seemingly prohibits even innocuous contracts. In a famous decision Justice Louis Brandeis ruled that the court must examine the restraint to see whether it "merely regulates and perhaps thereby promotes competition or whether it may suppress or even destroy competition."[8] This "rule of reason" is applied to balance the competitive benefits against the anticompetitive effects of an agreement. If the latter predominate, the restraint is unreasonable and, therefore, illegal. However, some restraints are blatantly anticompetitive and unjustified. These are "conclusively presumed to be unreasonable and therefore illegal . . . "[9] - or, *per se* violations.

> *The practice of medicine may be part art and part science; it is also "trade."*

There are four kinds of *per se* unreasonable restraints that competing physicians must avoid:

1. Price fixing - whether setting maximum or minimum fees, even if reasonable
2. Market division - allocation of consumers to different competitors
3. Concerted refusal to deal (group boycotts) - such as actions against insurers or denial of staff privileges to a competitor
4. Tying agreements - forcing buyer to purchase one service to get another

Apart from price-fixing, courts seem reluctant to apply *per se* treatment in health care cases, perhaps because the courts lack experience in characterizing these restraints as beneficial or not, or because the providers were acting in the public's interest.[10] Therefore, the rule of reason is applied. This does not mean the agreement is legal. It signifies only that the defendants have a chance to prove their actions are more procompetitive than anticompetitive.

The DOJ & FTC *Statements* describe "antitrust safety zones" for physician network joint ventures.[11] The number of physicians allowed in a network is limited to 20% (for an exclusive network) or 30% (non-exclusive) of the providers in each specialty in the geographic region. Exclusive networks are those where physicians do not have contracts except through the venture. The participating physicians must share "substantial financial risk" to qualify. This can be achieved with capitation contracts, compensation withholds, global fees or case rates, or any similar arrangements that indicate there is enough integration to achieve significant savings and efficiencies.[12]

The revised *Statements* permit physician networks that do not share financial risk. However, these ventures must: demonstrate sufficient clinical integration to generate significant efficiencies that benefit consumers; only use fee agreements reasonably necessary to attain these efficiencies; and, on balance, not be anticompetitive. The agencies will look for activities such as: utilization review and quality assurance programs; case management services; development of practice standards and treatment protocols; purchase of information systems to gather and evaluate data; and, hiring a medical director and staff.[13] This is likely to entail a large outlay of capital, more than most small groups can afford.

Physician ventures without the necessary financial risk-sharing or clinical integration are not likely to be approved. The agencies have considerable experience determining whether a venture exists merely to fix prices or otherwise block competition. There is no legitimate way for physicians to band together merely for contract negotiations. There must be a commitment to creating procompetitive efficiencies. Properly formed independent practice associations and group practices can negotiate for their members.[14] However, these ventures require a significant commitment in time and money.

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2. State law may also be relevant but is not discussed in this essay.
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7. Available at www.ftc.gov/reports/hlth3s
8. *Chicago Board of Trade v. United States*, 246 U.S. 231, 238 (1918).
9. *Northern Pac. Ry. v. United States*, 356 U.S. 1, 5 (1958).
10. Proger Philip A, et al. Health Care Networks and Managed Care: Antitrust Aspects of Integration and Exclusion § 03.A.2.d. (BNA's Health L. & Bus. Series No. 2500).
11. U.S. DOJ & FTC Statements of Antitrust Enforcement Policy in Health Care (Aug. 1996), (Statement 8, § A.) [hereinafter *Statements*].
12. Id. (Statement 8, § A.4.).
13. Id. (Statement 8, §§ B.1., C.1.).
14. The "messenger model" approved by the agencies and used by many preferred provider organizations cannot be used for negotiation; it is restricted to conveying contract offers between insurers and individual physicians, precisely because there is no integration of practices.

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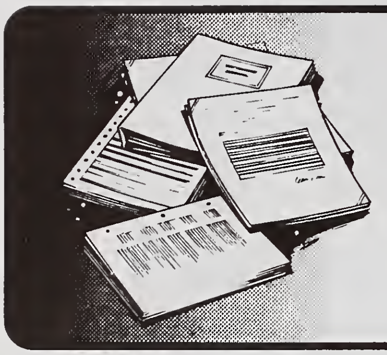

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A Multi-Center, Placebo-Controlled Trial of Melatonin for Sleep Distrubance in Alzheimer's Disease

Sponsor: Alzheimer's Disease Cooperative Study

Purpose: Brian Ott, MD, is conducting this trial to treat Alzheimer's Disease patients who experience sleep disturbances associated with Alzheimer's Disease. Two doses of melatonin will be used to treat sleep disturbances in order to lessen the burden on caregivers and family members.

Patients Recruited: Anyone 55 years or older with a diagnosis of probable Alzheimer's Disease experiencing sleep disturbances.

Intervention: Two doses of Melatonin vs. placebo

Duration of study: 12 weeks

Phase: III

Site: Alzheimer's Disease & Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860

Contact: Michael Pimental, MA, phone (401) 729-3752

Earlier vs. Later Levo-dopa in Parkinson's Disease (ELLDOPA)

Sponsor: NIH and the Parkinson Study Group

Purpose: Joseph Friedman, MD, is conducting this trial to compare the effect of early or late treatment of Parkinson's Disease with L-Dopa to answer the question of whether L-Dopa slows or hastens the progression of Parkinson's Diease.

Patients Recruited: Patients must be 30 years or older, must be diagnosed with Parkinson's Disease within the last 2 years. No eldepryl, L-DOPA, amantadine, anticholinergics, antihistamines, antidepressants, or benzodiazepines for previous 30 days.

Intervention: varying doses of Carbidopa/Levodopa vs. placebo

Duration of study: 9 months

Site: Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860

Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

A Phase III Multicenter, Double-Blind, Parallel-Group Placebo Controlled Study of the Effect of Riluzole 50 mg BID or 100 mg BID for Two Years on the Progression of Parkinson's Disease in 1050 Patients

Sponsor: Rhône-Poulenc Rorer

Purpose: The purpose is to treat recently diagnosed patients with the drug Riluzole, in order to study the effect this drug has on delaying the progression of Parkinson's Disease.

Principal Investigator: Joseph H. Friedman, MD

Patients Recruited: Patients must have recently diagnosed Parkinson's Disease with symptoms present for no more than 3 years and must currently be receiving no medications to treat Parkinson's Disease.

Intervention: Two doses of Riluzole or Placebo

Duration of Study: 2 years

Phase: III

Site(s): Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Margaret C. Lannon, RN, MS; phone: (401) 729-3750

Parkinson Disease Collaborative Study of Genetic Linkdate (PROGENI)

Sponsor: NIH and the Parkinson Study Group

Purpose: The purpose is to explore genetic, environmental and other factors which may play a role in the development of Parkinson's Disease.

Principal Investigator: Joseph H. Friedman, MD

Patients Recruited: Looking for sibling pairs (brothers and/or sisters) who both have Parkinson's Disease or suspected Parkinson's Disease. Siblings need NOT live in Rhode Island.

Intervention: None

Duration of Study: One visit

Site(s): Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Margaret C. Lannon, RN, MS; phone: (401) 729-3750

Psychosis and Associated Behavioral Disturbances in Patients with Dementia

Sponsor: a major pharmaceutical company

Purpose: The purpose is to determine whether persons with psychosis and behavioral disturbance related to Alzheimer's Disease or vascular dementia benefit from investigational antipsychotic drugs.

Patients Recruited: Patients must at be least 40 years old and have hallucinations or delusions associated with dementia caused by Alzheimer's Disease or stroke.

Intervention: One of two investigational antipsychotic drugs, or Placebo

Duration of Study: 28 weeks
Phase: III
Site(s): Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860
Contact: Michael Pimental, MA; phone: (401) 729-3752

A Randomized, Double-Blind Placebo-Controlled Trial to Evaluate the Safety and Efficacy of Vitamin E and Donepezil HC1 (Aricept®) to Delay Clinical Progression to Alzheimer's Disease (AD) from Mild Cognitive Impairment (MCI)
Sponsor: Alzheimer's Disease Cooperative Study and Pfizer/Elsai
Purpose: The purpose is to determine whether treatment of persons with mild forgetfulness can prevent the actual development of Alzheimer's Disease or slow the progression of Alzheimer's Disease
Principal Investigator: Brian R. Ott, MD
Patients Recruited: Patient must be 55 to 90 years of age and have memory difficulties that are not severe enough to interfere with carrying out one's usual daily activities (MMSE=24-30). Must be willing to take only vitamin supplement provided.
Intervention: Multivitamin, Vitamin E, or Donepezil HC1
Duration of Study: 3 years
Phase: III
Site(s): Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860
Contact: Michael Pimental, MA; phone: (401) 729-3752

Moving Forward
Sponsor: National Cancer Institute
Purpose: To examine the feasiblity of cancer survivors adopting a home-based exercise program. To evaluate the effects of home-based moderate exercise on: mood, physical symptoms (e.g., fatigue), weight, sleep, body image and quality of life.
Principal Investigator: Dr. Bernardine Pinto
Patients Recruited: Women ages 18 and over, diagnosed with stage 0-II breast cancer in the past 5 years.
Intervention: A 12-week home-based exercise program
Duration of Study: April 1998 - December 2002.
Site(s): The Miriam Hospital
Remuneration: Non-monetary incentive rewards (e.g., t-shirts)
Contact: Julie Parsons; phone: (401) 793-3851; fax: (401) 331-0335; e-mail: jparsons@lifespan.org

Pharmacotherapy of body dysmorphic disorder
Sponsor: National Institute of Mental Health
Purupose: Evaluate the effectiveness of psychotropic medication for body dysmorphic disorder, a distressing and impairing preoccupation with a nonexistent or slight defect in appearance
Principal Investigator: Katharine A. Phillips, MD
Patients Recruited: Age 18 to 65, both genders, no unstable medical problems, no history of seizures
Intervention: Patients receive a comprehensive psychiatric and medical evaluation. They are treated with medication which appears effective for body dysmorphic disorder. All evaluation and treatment are free of charge. For patients who participate in a placebo-controlled trial, patients randomized to placebo subsequently receive medication at no charge.
Duration of study: Three months
Phase: 4
Site: Butler Hospital
Remuneration for patients: None; however, all evaluations and treatment are provided free of charge.
Contact: Lisa Canuel; phone: (401) 455-6466; fax: (401) 455-6539; e-mail: Katharine_Phillips@brown.edu

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VITREO-RETINAL SPECIALIST

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BOOK REVIEWS

BOOKS BY RHODE ISLAND AUTHORS

Neuroscience: Exploring the Brain

Mark F. Bear, MD, Barry W. Conners, MD, and Michael Paradiso, MD
William and Wilkins, 1996

This volume is a suitable introductory textbook on the organization and function of the nervous sytem. Unlike most introductory textbooks, which target advanced undergraduate science majors, graduate students, and medical students, this text is written for students who may have a minimal knowledge of biology, physics and chemistry.

The book is divided into three parts: (I) Foundation, (II) Sensory, (III) Brain and Behavior. Part I introduces the modern field of neurosicence and traces some of its historical antecedents, ranging from Hippocrates to the 19th century German anatomist Otto Deiters. Inquiry into the nervous system began when "people were boring holes in each other's skulls (a process called trepanation), evidently not with the aim to kill, but to cure." The authors address how neuroscience research is conducted today, emphasizing the interdisciplinary approach. They examine the structure and function of individual neurons and their organelles, how neurons communicate chemically, and how cells form the nervous system. Chapters introduce the physiology of the neuronal membrane, interneural communication, modern methods used to study chemical synaptic transmission and the anatomy of the nervous sytem.

Part II examines the structure and function of the sensory (smell, taste, visual, auditory, somatic) and motor system. The authors use familiar examples to connect with students. Consider baseball: "strategies must be devised to move the body from the current state to one in which a pitch is delivered and the desired outcome is attained (a swing and a miss). The . . . (fast ball, curve ball, etc.) alternatives are filtered through the basal ganglia and back to the cortext until the final decision is made."

Part III tackles the neurobiology of human behavior, including mood, emotion, sleep, learning, memory, language and attention. The authors focus on the secretory hypothalamus, the autonomic nervous system, and the diffuse modulatory systems of the brain (ranging from electrical rhythms of the cerebrum during sleep and wakefulness) and disucss how various drugs and psychiatric disorders may disrupt these systems. They shift to the mechanisms employed during brain development that ensure that the correct connections are made between neurons. The authors concentrate on the anatomy and cellular mechanisms of memory and discuss the neuroscience of higher brain function. They discuss how progress is being made in understanding brain mechanisms of language and attention, concluding that it is not premature to begin contemplating the neurological bases of consciousness itself.

While a medical student may require additional clinical references, this text is not intended for such advanced students. The main strength of this readable book is that it combines common sense metaphors, real-world examples, humor and anecdotes to inspire elementary science and nonscience students with a fascination for scientific facts, method, and discovery. The authors helpfully provide chapter outlines, a neuroscience glossary, review questions at the end of each chapter, selected additional readings, beautiful illustrations by John Woolsey, and special interest boxes dealing with topics ranging from multiple sclerosis to magnetic resonance imaging. The authors' stories are scattered throughout the text as "Paths of Discovery" boxes, which point out the "accidents" behind discoveries and emphasize the importance of patience and industry in reserach. In short, this intriguing introduction to the field of neuroscience not only teaches the elementary student effectively, but more importantly, shares the human side of neuroscience.

– Neetha Shetty, AB

Neetha Shetty is a fourth-year student in the Brown University School of Medicine.

Neurology in Primary Care

Joseph H. Friedman, MD
Butterworth & Heimann 1998

Neurology in Primary Care by Dr. Joseph Friedman is an informative and highly readable book which allows the primary care physician access to the often elusive world of the clinical neurologist. In clear and concise prose, the author highlights common clinical problems in neurology. Each chapter begins with a description of a particular neurological syndrome, followed by a discussion of the pertinent historical and physical exam findings. The author then discusses the differential diagnosis of the syndrome and the treatment strategies, concluding with helpful clinical pearls. In addition, each chapter highlights worrisome symptoms which require more attention and possible neurologic studies, including imaging.

The book is not meant to serve as a reference text, but rather to be read in its entirety or selectively, by a particular syndrome of interest. The book devotes little space to pathophysiology, except as a guide to understanding the signs and symptoms of a unique clinical entity. Dr. Friedman gives the reader insight into neurologic questions that all primary care physicians ponder; e.g., when to consider stopping anticonvulsants; the appropriate time to neuroimage the brain in a patient with a headache; and the "red flags" for serious causes of back pain. Additionally, he outlines the clinical problems that should be referred to the neurologist, providing reassurance to the primary care physician seeking to confirm his or her patient management.

Throughout each chapter, the reader profits from the author's extensive clinical experience. The conversational style of writing makes the reader feel that s/he is engaging in the highest quality neurology rounds. The author focuses on a particular problem, highlighting all its salient clinical features and invoking evidence-based medicine when necessary. Beyond the academic discussion, he fully appreciates the psychosocial impact of certain neurological conditions. For instance, in the chapter on memory loss, cognitive dyssfunction, and mental status changes, he thoughtfully examines the impact of Alzheimer's disease on the patient and family. A consistent message is the author's enthusiasm for neurology and teaching, as well as his compassion for patients.

The appendices on the neurological exam and neurologic tests are as packed with helpful information as the chapters. Overall, this book is an extremely useful source of practical information for primary care physicians, as well as for medical residents or students rotating on a clinical neurology inpatient or consultation service. The book complements the knowledge of the internist attending on the general medical wards. It is small enough to be carried in the pocket of a lab coat. Every practicing generalist could reinforce and enhance his/ her clinical acumen in neurology by reading this wonderful book.

– Kelly McGarry, MD

Kelly McGarry, MD, is attending internist, Rhode Island Hospital, and Associate Director of the General Internal Medicine Residency Program, Rhode Island Hospital.

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Seated: Dr. Nicholas Iannuccilli; Standing, left to right: Dr. Kathleen Staudinger, Dr. Charles Rosenthal, Dr. Karen Ackerman and Dr. Daniel Druckman; Not pictured are Dr. Paul Hessler and Dr. Rodolfo Zaldivia

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Handbook for Prescribing Medications During Pregnancy

Donald R. Coustan, MD and Tara K. Mochizuki, Pharm, JD
Lippincott Williams & Wilkins, 1998

Let me begin by saying "Hurray" to Dr. Coustan and Tara Mochizuki for the Third Edition of *Handbook for Prescribing Medications During Pregnancy*. It has been eagerly awaited.

The book is an updated desk top and portable reference. It gives an important and thorough approach to the use of medications, including recommendations, contraindications, and safety. The book gives a synopsis of current relevant information about the effect of many individual drugs on the pregnant woman, her fetus and breast feeding.

Each entry begins with indications for use of a particular drug, and the authors' specific recommendations. It supplies information on dosage, general adverse effects, pharmacological properties, mechanisms of action, biotransformation and references on the medications used in pregnancy. Contradictions, both absolute and relative, are discussed for all drugs; and other therapeutic options for certain medical situations are also discussed and outlined.

It is a very practical and thorough book on the use of many different medications, from over-the-counter medications and commonly used drugs to uncommon and rarely used drugs in pregnancy, such as oncology medications and antivirals. The reference also addresses issues of inadvertent exposure in early pregnancy and illicit drug use. It gives the practioner a sense of security regarding medication prescribing during pregnancy.

The day I was asked to review the book, I received two phone calls: one from a family practioner and one from the post-op recovery room regarding certain medications in pregnancy and lactation. I felt very comfortable and secure after consulting this *Handbook*.

The book also discusses important controversies and clarifies use of medications in pregnancy including steroids, anesthesia, fertility agents and new medications.

It is user-friendly, making it easy to use as a reference.

Thus, in summary, I am ecstatic regarding this release of the Third Edition. It has been long awaited. We salute Drs. Coustan and Mochizuki for a thorough and accurate evaluation of medications used in pregnancy. It was a tremendous amount of work and research, and I give them much credit.

– Patrick J. Nugent, MD

Patrick J. Nugent, MD, is Obstetrician-in-Chief, Memorial Hospital of Rhode Island, and Clinical Assistant Professor, Brown University School of Medicine.

Prognosis in Neurology

James Gilchrist. MD
Butterworth Heinemann,1998

There is something for everyone in *Prognosis in Neurology*. Medicine has made and continues to make astonishing advances. However, as we treat patients, this progress often seems all too limited. Prognosis is and will continue to be a primary concern to clinician and patient alike until the unlikely day arrives when all illness is easily cured. James Gilchrist states in the preface, "Many patients' first question after the examination is 'What is going to happen to me?'" Dr. Gilchrist, along with 94 contributing authors (22 are from Rhode Island and no doubt many more have a connection to our community), set out to answer that question for 81 disorders or classes of disorders. In doing so, they have created an excellent general text of neurology, focusing on prognosis.

Dr. Gilchrist did an effective job of bringing consistency throughout *Prognosis in Neurology* despite the diversity of his contributors and the disorders addressed. Each chapter consists of an introduction that typically describes the disease and its epidemiology. The natural history is then addressed, followed by sections labeled "Factors Affecting Prognosis," "Evaluation for Prognosis," and "Therapies Affecting Prognosis." Short and long-term prognoses are then presented with a bibliography of additional reading. Each topic is allocated approximately 4 pages.

This organizational structure succinctly presents a description of the disorder, appropriate therapies and prognosis. Differential diagnosis of syndromes is critical to determining prognosis and disease subtypes are critical to determining prognosis; therefore, when necessary, diagnostic considerations are also addressed. On occasion, Dr. Gilchrist appropriately allows an author to briefly stray when the topic addressed is a syndrome or subject encompassing multiple diseases and the format is too constricting.

There are times when the format can be somewhat contrived. For example, the chapter on Alzheimer's Disease addresses short-term prognosis. There really is no expectation for short-term changes but Dr. Brian Ott very reasonably addresses near-term management. An author writing on Meningeal Carcinomatosis acknowledges that "long-term" is rarely even a year. Other authors occasionally push the category boundaries in a way that brings them slightly off the path we expect.

Categories of disease such as disorders of consciousness, epilepsy, degenerative disorders, neoplastic disease, infections, and developmental disorders result in an extensive array of subjects of interest to physicians in primary care, adult medicine, pediatrics, and neurology. Dr. Gilchrist risked creating a collection too basic for the neurologist and too focused for the generalist; however, the skill of his editing and the work of his contributors resulted in a text of broad interest and significant utility.

– Peter Hollmann, MD

Peter Hollmann, MD, is Medical Director, Coordinated Health Partners, and Assistant Clinical Professor of Medicine, Brown University Medical School. He is also a member of the editorial board of Medicine & Health/Rhode Island.

Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	April 1998	12 Months Ending with April 1998		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	241	3,218	325.0	4,032.5
Malignant Neoplasms	218	2,510	253.5	7,211.5
Cerebrovascular Diseases	52	692	69.9	717.0
Injuries (Accident/Suicide/Homicide)	18	320	32.3	6,163.5**
COPD	30	470	47.5	335.0

Vital Events	Reporting Period		
	October 1998	12 Months Ending with October 1998	
	Number	Number	Rates
Live Births	1,229	13,231	13.4*
Deaths	781	9,818	9.9*
Infant Deaths	(6)	(104)	7.9#
Neonatal deaths	(5)	(82)	6.2#
Marriages	951	7,606	7.7*
Divorces	211	3,203	3.2*
Induced Terminations	386	4,698	355.1#
Spontaneous Fetal Deaths	65	972	73.5#
Under 20 weeks gestation	(60)	(896)	67.7#
20+ weeks gestation	(5)	(76)	5.7#

* Rates per 1,000 estimated population # Rates per 1,000 live births

**Excludes three deaths of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 990,225

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

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The Official Organ of the Rhode Island Medical Society
Issued Monthly under the direction of the Publications Committee

VOLUME 1
NUMBER 1

PROVIDENCE, R.I., JANUARY, 1917

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NINETY YEARS AGO

[APRIL, 1909]

In "Medical Supervision of Schools and Its Relation to the General Practitioner," J. H. Akers, MD, complained that "medical supervisors," spotting children with enlarged tonsils and/or adenoids, did not contact the family's general practitioner, but instead referred those children directly to a specialist at the hospital. The mother of one of his patients came with her son to the office, carrying the "official" card that directed, "Go to the Outpatient Department of the Rhode Island Hospital, Nose and Throat Department, on Monday, Wednesday or Friday, between 9 and 10" for the operation." In another case, a nurse sent by the Health Board "... stated to the mother...that the enlarged tonsils and adenoids would press on the child's brain and so cause her son to be an idiot, fast and sure, at the age when he should be of assistance to the family."

Although conceding that some children needed the operations, Dr. Akers noted that he could do the operations at home. Also, although some families couldn't afford medical care, Dr. Akers cautioned, "Perhaps if we could see the bank books of some who get this assistance and compare them with many of our physicians in the city, we might be better able to decide who the poor really are."

Charles Chapin, MD, Superintendent of Health, Providence, reported 332 deaths for March (annual rate: 18.05/1000) - 23 fewer than March 1908. Consumption was the key cause (82 deaths). In February and March, Dr. Chapin tabulated 75 cases of scarlet fever(1 death), 78 cases of diphtheria (8 deaths), and 9 cases of typhoid fever. There was "very little" measles or whooping cough, and no case of rabies, during those months.

FIFTY YEARS AGO

[APRIL, 1949]

Captain Charles F. Behrens, MC, USN, discussed "Radiologic Safety in Atomic Warfare," (from a presentation at the Joint Meeting of the Rhode Island Medical Society and the Providence Medical Society at Quonset Port): "Fundamentally ionization alters important chemical compounds, tends to inactivate enzymes, disrupts genes, and causes abnormalities in the chromosomes..." He conceded there was "no accepted specific treatment" and "up to a hundred thousand or more casualities may occur from a single bomb."

Jess P. Eddy, III, MD, in "Sympathectomy: Its Role in the Treatment of Vascular Disease," tabulated that of 1269 deaths in Providence, 440 had coronary injuries, 255 cerebral accidents, 212 had arteriosclerotic heart disease. "The operation of sympathectomy is... not a curative procedure, but by...encouraging the development of collateral circulation, it goes a long way toward alleviating suffering."

An editorial bemoaned the focus on inpatient training: "Our interns and residents prepare themselves for practice by handling the most spectacular of our diseases. These diseases are often spectacular because they are few in number. It must be a rare young man who starts in practice handling largely that type of case....The trainee should see the colds, the coughs, and the indigestions, the aches and pains, that bring people to our waiting rooms."

TWENTY FIVE YEARS AGO

[APRIL, 1974]

Stanley M. Aronson, MD, in "A Message from the Dean," urged a state-wide bloodbanking system. "Since blood is a substance which cannot be synthesized but requires voluntary donation and since it is a fluid which cannot be stored beyond approximately 4 weeks of refrigeration, it has become increasingly apparent that individual hospital-based blood banks operating without some system of integration, are at the mercy of a 'feast or famine' blood economy." That coordination had begun. Under Dr. Herbert C. Lichtman (Miriam Hospital), the blood banks of Cranston General, St. Joseph's Hospital, Roger Williams General Hospital, Fogarty Memorial Hospital, Notre Dame Hospital, Woonsocket Hospital, Veterans Administration Hospital, Rhode Island Medical Center, and Miriam coordinated blood collection and sharing (the Community Association Blood Banks of Rhode Island). Thus far, in Rhode Island the purchase of blood from commercial sources had been "virtually eliminated."

Robert S. Burroughs, MD, alerted readers to a patient with Munchausen's syndrome. The 29 year-old former paramedic, "well known in Boston for the last 2 years," ususally presented "with hematuria and flank pain." He was recently discharged from Memorial Hospital.

Seebert Goldowsky, MD, Chair-elect, Rhode Island Interagency Council on Smoking (and Editor-in-Chief, *Rhode Island Medical Journal*), testified before the State Senate on behalf of Bill S2212: An Act Prohibiting Smoking in Public Places: "Nonsmokers have certain rights to health to which very little attention has been paid." A related editorial gave smoking statistics for hospitals in Rhode Island: 75% of hospitals let patients smoke in their rooms (even double rooms); 90% let visitors smoke in patients' rooms; 30% let employees smoke in areas where there were patients.

In his Annual Address, Thomas A. Head, MD, President of the Providence Medical Association, decried the misplaced enthusiasm for large group practices, citing the following HEW costs-per-patient-visit: $21.16 in a health center, $19.00 in a group practice, $9.59 in a private practice.

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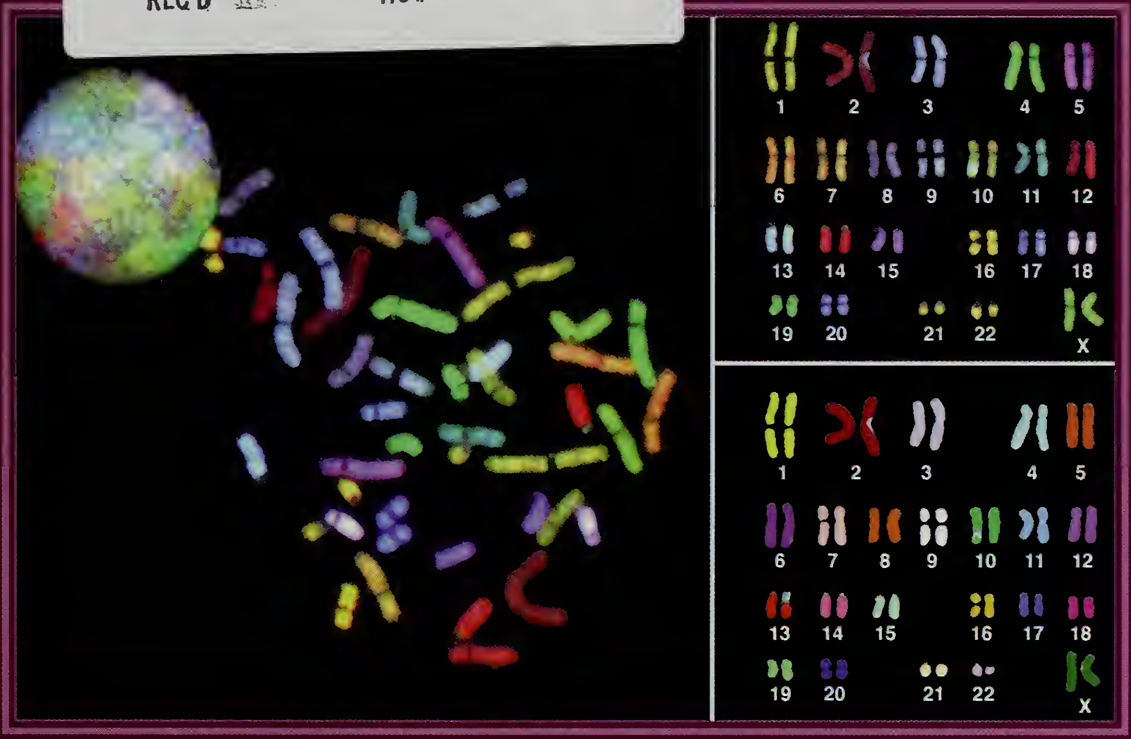
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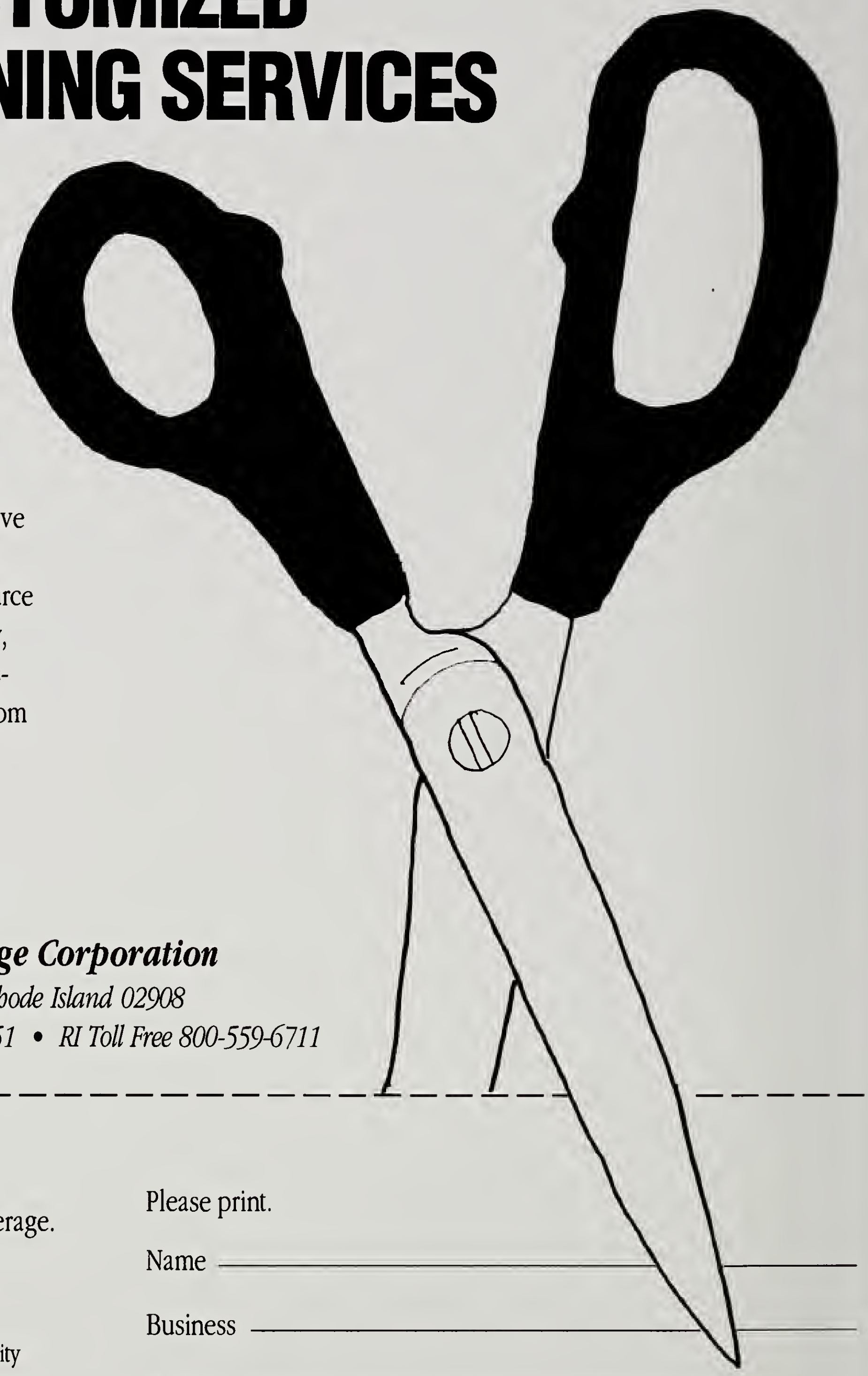

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COMMENTARIES

Trojan Horse Advertising

As a medical educator I have been increasingly impressed by pharmaceutical companies' efforts to sway our opinions through main-line academic journals. Recently I received an unsolicited newsletter published by a "health system," incorporated, of course, reviewing "new trends in prescription therapies, benefits and costs" in depression. Although I'm a neurologist, I see many depressed patients and prescribe a fair amount of the drugs discussed. I try to find out who sponsors these unsolicited Trojan horse ads to see if there's a potential bias since the publisher's name never provides a clue. The publisher is always something like "Educational Forum," "Health News, Inc.," "Modern Management," etc. This particular notice was from "an information driven health solutions company," God help us, which is a subsidiary of a large drugstore chain, underwritten by a grant from a pharmaceutical company, whose drug was well reviewed in the leaflet.

I use this drug. I think it's a good drug. And perhaps more importantly, I liked the three page flier. It read like a concise journal article and may well have provided an honest appraisal of the drugs at hand. On the other hand, I'm not an expert in the field and knowing that this publication was subsidized by an interested party raised my level of skepticism. In fact, it put me over the top and the review/ad (infomercial) has undermined itself. In response to my revelations, I must assume that all comparisons are biased, and so I take away little from the reading, perhaps feeling a bit suspicious and less likely to use the recommended drug. How this works on my unconscious, of course, I have no idea.

This leads to another consideration, possibly a more sinister form of subliminal advertising, the journal supplement. In many medical journals the release of a new drug is marked by the publication of a special supplement devoted to marketing this drug in one or more appropriate specialty journals. In neurology, for example, the new Parkinson's disease and epilepsy drugs have spawned, "Introducing a New Paradigm," and other self-aggrandizing titles for supplements that devote themselves to one topic in which this new drug figures prominently. The supplements are guest edited and the papers are typically written by world experts who render up-to-date, honest opinions. The journal publishers clearly state their lack of oversight and just as clearly indicate where the funding comes from. You will never see such a supplement containing an article stating, "this drug is a dog." The drugs always seem great because only enthusiastic authors are recruited. They are not "bought." Their *choice* was biased. Unwary readers may simply assume this work is just the same as the work contained in the anonymously peer-reviewed journal, which it is not. The unwary reader skims the abstracts and sees how great this drug is in a professional, non-commercial format. One recent supplement devoted to a drug dwelt on the theoretical benefits of this drug whereas the one article on the drug itself noted that the five-year study found no benefit over the cheaper, generic, shorter acting version. On the one hand this is honest while on the other a quick review by the non-super specialist creates a different viewpoint.

Why do journals do this? For money. These supplements subsidize the main journal. Subscribers and specialty association members would have to pay increased fees. My own recommendation is to print the pharmaceutical sponsored supplement on differently colored paper, boldly stating that the costs of publication are underwritten by a grant from Drug Company X whose product is herein reviewed.

Health care professionals are bombarded by a lot of information, and much of our education comes from drug reps. We know this information is biased. We know the ads we see are biased. I worry that not enough of us recognize the subtle biases of these other "subliminal" or "Trojan horse" advertising.

– Joseph H. Friedman, MD

ON THE COVER Ordinarily *Medicine & Health/Rhode Island* features the work of artists. In this instance, the human chromosome represents so dramatic a configuration that it deserves to stand alone as a work of art.

He Hath the Falling Sickness

Few diseases arise so abruptly, speak so boldly, and define themselves so readily, as epilepsy. There is little clinical subtlety when the major form of epilepsy descends upon its victim. The unbidden affliction announces itself explosively with altered consciousness, inarticulate cries and diminished motor control, convulsions and incontinence followed by an amnestic interval of variable length.

The early Greeks called it *Herakliea nosos,* the illness of Hercules, since the legendary hero had been prone to seizures. Most commonly, however, people called it the falling sickness. The Hebrews, too, named it "the falling down" or, "one who writhes."

There are scattered references in the Scriptures to spontaneous falling down, with the victim sometimes entering a trance with his eyes open [Num 24:4.] Saul, King of the Israelites, was said to have suffered from seizures. But it awaited the apostolic writings of Mark, Luke and Matthew for a narrative description of the disease. Chapter 9 of Luke begins with Jesus assembling his twelve disciples and giving them "power and authority over all devils, and to cure diseases." And this story is then told: "And, behold, a man of the company cried out, saying, Master, I beseech thee, look upon my son: for he is my only child. And, lo, a spirit taketh him and he suddenly crieth out; and it teareth him that he foameth again, and bruising him hardly departeth from him. And I besought thy disciples to cast him out; and they could not." It is a poignant story, easily understood by any diligent parent. And it describes a disease that will not respond readily to casual therapies.

Ancient Rome had intimate knowledge of epilepsy. They called it by various names including *morbus caducus* [the falling sickness]; *morbus comitialis* [disease of the assembly hall.] It was a standing Roman custom to shut down the public assembly [*comitia*] for ritual purification whenever any legislator experienced a seizure; *morbus sacer* [the sacred sickness]; or *morbus demoniacus* [the demonic sickness.] In Act 1, scene 2 of Shakespeare's *Julius Caesar,* Casca recounts one of Caesar's many epileptic attacks: "He fell down in the market-place, and foamed at the mouth, and was speechless." Brutus responds: "'Tis very like, he hath the falling sickness." Indeed, epilepsy seemed to have been so common amongst the leaders of antiquity [eg, Caesar, Alexander the Great, even Caligula] that some thought it to be a necessary prelude to military greatness.

Both educated and uneducated have viewed this disease with horror: Many believed that the victim's body had been possessed and its control relinquished to some outside agency. A few believed that this possession was from divine sources and accordingly they rendered respect, even homage, to the epileptic; but most contended that the possessing forces were Satanic therefore requiring that they be displaced by theological rather than medical intervention. John of Gaddesden, English court physician of the 14th Century, advised that the physician whisper into the ear of the epileptic: "Depart demon and go forth !" He commented further, "This species of devil is not cast out save by prayer and fasting. The patient should then write out this gospel and wear it about his neck and he will be cured."

Yet it was Hippocrates, writing on the island of Kos some 1,700 years earlier, who declared that epilepsy was not a spirit-possessing disease but a disorder much like any other: "And they who first referred this disease to the gods, using the divinity as a pretext and screen of their own inability to afford any assistance, have given out that the disease is sacred. . . Neither truly do I count it a worthy opinion to hold that the body of man is polluted by God."

Epilepsy was a disease which also plagued many British leaders including Alfred the Great, William III and William Pitt. Many of Queen Victoria's relatives had been epileptic and she provided funds for the National Hospital for the Paralyzed and Epileptic, in London, so that research into the cause and care of the epileptic might be furthered.

Epilepsy seemed to have been no hindrance to genius. The ranks of those prone to seizures included: Dickens, Moliere, Handel, Schumann, de Maupassant, Byron, Flaubert, Dostoyevski, Swinburne and van Gogh. Many writers incorporated their own clinical episodes into the substance of their novels. No medical text equals the sense of authenticity exhibited in Dostoyevski's vivid fictional descriptions of epileptic seizures.

Prior to the 19th Century, therapy varied from the bizarre to the ineffectually innocent. The blood of gladiators or executed prisoners was frequently recommended as a preventive measure; and Hans Christian Anderson recalls seeing parents force their epileptic children to drink the warm blood of recently beheaded criminals. A standard anti-epileptic decoction consisted of the boiled and macerated leaves of elder, garlic, peony and mistletoe. The stated rationale for the use of mistletoe was that it tenaciously clung to the upper branches of the oak tree never falling to the ground and hence must be helpful in the falling disease.

The 19th Century physician, baffled and humbled by epilepsy, believed that it was of unknown cause, inscrutable and, alas, incurable. But gradually the neurophysiologists explored the nature of normal brain impulses and the manner in which certain portions of the cerebrum held dominion over motor activity in the limbs. Epilepsy was then conceived of as an explosive happening in which ungoverned impulses were initiated from an abnormal cerebral locus. The idea of supracorporeal possession was now replaced by the more secular concept of abnormal excitation arising from a pathological segment of cerebral cortex. And gradually, too, effective therapies such as the bromides, barbiturates and dilantin were discovered.

This current century has witnessed significant inroads in the understanding, diagnosis, treatment and even prevention of the epilepsies. Studies have determined that there are many forms of epilepsies: some awesomely dramatic such as the *grand mal* seizures; but others with neither convulsive movements nor other visible abnormalities; and with, at most, a brief spell of detached staring [*petit mal*.] The epileptic diseases have been shown to be caused by a spectrum of unrelated ills including birth injury, toxins, metabolic insults, vascular disorders, heritable neurological lesions, even brain tumors. And current treatment has evolved to a stage where the great majority of epileptics may now lead productive lives unburdened by the threat of seizures. But society's memory is long; and there are still those who think of epilepsy as possession by demonic forces.

– Stanley M. Aronson, MD

Medical Genetics: Past, Present and Future

Dianne Abuelo, MD

EARLY HISTORY OF HUMAN GENETICS

From a historical point of view, knowledge of human genetics has its roots in antiquity; the ancient Hebrews were aware of the natural history and mechanism of inheritance of the disease we now call hemophilia, as they omitted the obligatory ritual circumcision in male infants whose brothers or maternal uncles had bleeding disorders. Many centuries later, in the mid-1800s, Gregor Mendel deduced the basic rules of inheritance from his experiments on garden peas; before that time, inheritance was thought to be a merging of characteristics of both parents. Mendel realized that there were discrete units of heredity which were passed to offspring in a predictable manner. Unappreciated until early in this century, his observations became the cornerstones of modern genetics when the inheritance of inborn errors of metabolism was studied and found to fit his rules.

MODERN HISTORY OF MEDICAL GENETICS

As a medical subspecialty, genetics is quite young; knowledge necessary for its recognition began to accumulate in 1956, when we learned that the normal number of chromosomes in humans is 46, not 48 per cell, as had been previously thought. Geneticists discovered the chromosomal origins of Down syndrome, Turner syndrome and Klinefelter syndrome during the next few years. Watson and Crick deciphered the genetic code in the 1960s, and around that time, prenatal diagnosis by amniocentesis was developed. Techniques for differential staining of chromosomes led to the mapping of genes on their chromosomes during the 1970s, and cloning of human genes began in the 1980s. In this decade, new molecular technologies have elucidated the actual structure of many disease-causing genes and, with that knowledge, the ability to test patients for these genetic disorders has increased markedly.

New mechanisms for genetic disorders that do not follow Mendel's rules have recently been identified; for example, trinucleotide expansion syndromes (from repetitive stretches of DNA triplets) that cause Fragile X Syndrome, Huntington's disease, and several other CNS disorders. Microdeletions (tiny missing pieces of chromosomes detectable with fluorescent techniques) can lead to Prader-Willi Syndrome and interestingly can cause altogether different syndromes, depending on whether the microdeletion has been inherited from the father or the mother (referred to as genetic imprinting).

Abbreviations Used:

CF	cystic fibrosis
ELSI	Ethical Legal and Social Implications
FISH	fluorescent in situ hybridization
PCR	polymerase chain reaction
PKU	phenylketonuria

The Human Genome Project, a coordinated international initiative, will produce detailed maps of each human chromosome and determine the base sequence of all approximately 100,000 human genes. Molecular geneticists are discovering new techniques as part of this endeavor, and the Project is running ahead of schedule. One arm of the Project focuses on the Ethical, Legal and Social Implications (ESLI). Interestingly, Frances Alexakos surveyed Rhode Island primary care physicians, asking their attitudes on ordering genetic testing for breast cancer. Those results, abstracted in this issue, indicate strong interest in testing as well as concerns about informed consent, emotional stress and medical-legal considerations. Public education is also a priority of the Genome Project. The above endeavors should allow individuals to make informed decisions on their health and reproduction. Results are also expected to enhance the practice of medicine in general, and more specifically, the development of gene therapy in the future.

GENETICS IN SUBSPECIALTIES

Clinical geneticists care for patients with various types of birth defects and genetic disorders. Most activity currently takes place in academic departments of pediatrics and obstetrics. However, genetic knowledge has begun to diffuse into other subspecialty areas of medicine. For instance, gastroenterologists who care for patients with familial polyposis have traditionally offered at-risk family members surveillance procedures, including periodic colonoscopies. In many families, DNA testing can determine which relatives have inherited the causative gene, thus predicting which individuals require prophylactic colectomies and which ones will be free from the disease. Developmental pediatricians can now diagnose Fragile X syndrome, the most common cause of inherited mental retardation, by means of a DNA test, which can also be used by genetic counselors to diagnose both female and male carriers of Fragile X. Neurologists can diagnose an ever increasing number of disorders by DNA analysis, including Duchenne muscular dystrophy, myotonic dystrophy, Huntington's disease, etc. Obstetricians perform amniocenteses for a wide variety of genetic indications. They request screening for Tay Sachs carriers in their pregnant patients of Ashkenazic Jewish or French-Canadian ancestry; population-based screening for carriers of Tay-Sachs has dramatically reduced the incidence of this lethal disease. Craniofacial surgeons operate on children and adolescents with birth defect syndromes, some of which can now be specifically diagnosed with genetic techniques. Even consultants in child protection can sometimes benefit from availability of collagen testing to help differentiate physical abuse from genetic types of brittle bone disease. Forensic DNA testing is used for more accurate identification of criminal suspects and conversely, for protection of the falsely accused.

> *Rhode Island is the first state in the United States to institute universal hearing screening in the newborn period, resulting in opportunities for early amplification and better prognosis for language acquisition.*

Screening Newborn Infants for Congenital Disorders

Modern laboratory capabilities offer the potential ability to screen members of a population to ascertain those individuals at high risk of either inheriting a genetic disorder or producing affected offspring. In obstetrical medicine, this involves techniques to identify patients from the general pregnant population who are at high risk for having infants with chromosomal disorders or neural tube defects, in order to offer genetic counseling and further diagnostic procedures. In pediatric medicine, this involves identifying babies from the normal newborn population who can benefit from specific therapy.

Metabolic disorders, in past years, were important causes of infant mortality as well as morbidity in the form of mental retardation in the survivors. Before the mid 1960s, patients affected with phenylketonuria (PKU) appeared perfectly normal at birth, but gradually developed behavior problems, seizures and mental retardation and were frequently institutionalized. In 1953, a breakthrough in biochemistry led to the discovery that an enzymatic defect prevented the metabolism of phenylalanine, a component of dietary protein. With this knowledge, clinicians learned to treat affected individuals with a special diet low in phenylalanine. Detection of the disorder in newborns and appropriate dietary management resulted in a dramatic improvement in prognosis; persons with PKU can now be expected to attain normal intelligence, longevity and quality of life.

The Rhode Island Department of Health supervises the screening of all newborn infants not only for PKU, but for several other treatable metabolic disorders as well as congenital hypothyroidism, which can be treated with replacement therapy before irreversible damage occurs. Peter Simon, MD, MPH, James Sullivan, PhD, and Ronald Caldarone, MSW, outline the Rhode Island Newborn Screening Program, including a brief description of congenital adrenal hyperplasia, which can result in ambiguous genitalia. Affected newborns can develop severe electrolyte disturbances and dehydration if not detected and treated expeditiously. We now screen for hemoglobinopathies, since prophylactic antibiotics have been shown to decrease mortality due to infection in sickle cell disease. We recently have begun to screen babies for hearing loss; Rhode Island is the first state in the United States to institute universal hearing screening in the newborn period, resulting in opportunities for early amplification and better prognosis for language acquisition.

Laboratory Genetics

In Rhode Island, there are several laboratories and clinical centers with expertise in the diagnosis and treatment of genetic disorders, a sampling of which are described in this issue. Clinical genetics laboratories are essential for confirmation of diagnoses of individuals with well-known conditions, such as Down syndrome, in which an extra chromosome is present. However, many patients present with varying combinations of birth defects, dysmorphic features and mental retardation and do not have an easily recognizable syndrome. A combination of molecular and cytogenetic methods can now diagnose many of these indi-

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viduals. Umadevi Tantravahi, PhD, (Women & Infants Hospital) and Hon Fong Louie Mark, PhD, (Rhode Island Hospital) illuminate some of these new methods, including Fluorescent In Situ Hybridization (FISH), Southern Blot and polymerase chain reaction (PCR).

CYSTIC FIBROSIS

Mary Ann Passero, MD, discusses the most common serious inherited disorder in the Caucasian population, cystic fibrosis (CF). Advances in treatment of CF have improved the outlook for affected individuals considerably, with the average life span increasing from approximately 14 years to 31 years since 1969. The molecular basis of CF is now known, but interestingly, the diagnosis of CF can still be problematic. Affected individuals in Rhode Island receive coordinated, multidiscipinary care, including genetic counseling, at the Cystic Fibrosis Clinic of the Rhode Island Hospital.

Prevention of CF, by carrier detection and prenatal diagnosis is the subject of much current controversy. The American College of Medical Genetics currently recommends that "couples in which one or both reproductive partners either has CF or has an affected relative should be offered carrier testing and counseling," but that "it is not yet appropriate to offer genetic testing for CF to the entire adult population, including couples pregnant or planning a pregnancy." The College is concerned that the infrastructure for providing proper educational and counseling services is not yet in place for such an endeavor.

GENETIC COUNSELING AND HUNTINGTON'S DISEASE

Master's degree level genetic counselors are trained to counsel and educate genetics patients and have become an indispensible part of genetic health care. Rhode Island Hospital, the Prenatal Diagnostic Center and the Breast Health Center of the Women & Infants Hospital employ genetic counselors. Amanda Walsh, MS, describes the role of genetic counseling in caring for at-risk family members of patients with Huntington's disease. This is a particularly challenging endeavor, as relatives have usually witnessed the devastating effects of this disorder, i.e. the gradual neurologic deterioration of their family members. Individuals frequently have mixed feelings as to whether they really wish to know if they have inherited the causative gene and may also have legitimate concerns about their risks to incur genetic discrimination in employment and health insurance.

CANCER GENETICS

All cancer is genetic, i.e. caused by mutations in certain genes. In most cases, the mutation or mutations have been random events that began in a somatic cell. However, a minority of forms of cancer are hereditary, since the initial causative mutation is carried in all cells, including the gametes, and is therefore capable of being passed from one generation to another.

Some of these genes have been discovered, including two that cause cancer of the breast, BRCA1 and BRCA2. Robert Legare, MD, describes current screening, counseling and management for affected women.

GENETICS ON THE INTERNET

Reflecting changes in recent knowledge, the Internet has abundant resources for patients as well as physicians. The definitive catalog of genetic diseases, *Mendelian Inheritance in Man,* by Dr. Victor McKusick, is available free of charge online at http://www3.ncbi.nlm.nih.gov/Omim/. Helix, which is a directory of DNA laboratories in the United States and around the world is available to qualified medical professionals at http://healthlinks.washington.edu/helix/. The Alliance of Genetic Support Groups Directory has information for families on many common and rare conditions at http://medhlp.netusa.net/agsg/agsgsup.htm and the Gene Letter at www.geneletter.org/ covers new scientific discoveries and explores the ethical issues surrounding them.

CONCLUSION

Genetic medicine is no longer an esoteric specialty; its concepts and technologies can apply to all branches of medicine at present and it will doubtless become yet more influential in the future. The articles in this issue of *Health & Medicine, Rhode Island* provide some basic information for medical professionals, who are encouraged to call on any of us for help and discussion for any specific questions they might have.

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I am pleased to acknowledge the assistance of Peter Lee, a fourth-year Brown University MD/PhD student, in assembling this special issue on genetics. Peter contributed energy, talent, and insight; I look forward to seeing his name on additional scholarly endeavors in the years ahead.

– Dianne Abuelo, MD

Newborn Screening Program in Rhode Island: an Update

Peter Simon, MD, MPH, James Sullivan, PhD, Ronald Caldarone, MSW

Abbreviations Used:

PCP	primary care provider
PKU	phenylketonuria
RIDOH	Rhode Island Department of Health

One of the most satisfying aspects for all working in maternal and child health is the overwhelming statistic predicting that almost 95% of newborns will be discharged healthy. From time to time, however, a healthy infant in an asymptomatic phase of one of eight genetic conditions will be detected. The following scenario reflects what should happen in Rhode Island.

A mother and baby are continuing normal postpartum trajectories at home after discharge on the second day of life. A call from the Massachusetts Department of Public Health Laboratory is received, on the child's sixth day of life, at the Rhode Island Department of Health Laboratory by a senior chemist in the newborn screening lab. Massachusetts reports an abnormal 17-ketosteroid level on a screening specimen obtained by the birthing hospital laboratory on day two of life, prior to hospital discharge. The senior chemist pulls up the infant's laboratory record on the computer and identifies the primary care provider (PCP) at the time of hospital discharge and confirms this information and other family information through an electronic link with the Division of Family Health's KIDSNET database. Next, the chemist calls the PCP to ask that the family be immediately referred to the Endocrine/Metabolic Division of the Pediatric Department at Hasbro Children's Hospital to schedule a diagnostic assessment that will confirm the elevated screening test for congenital adrenal hyperplasia. The PCP, who gets prior approval from the family's Managed Care Organization, arranges the screening. (Under RI General Law, insurers must pay for all necessary services required to diagnose and treat this genetic condition). The endocrine fellow on call meets the family in the Emergency Department and finds an infant with a recent history of poor feeding, decreasing numbers of wet diapers and one episode of vomiting. On exam, the baby is 400 grams under birth weight, has dry mucous membranes and other than absence of testes in the scrotum appears normal. Laboratory studies on blood and urine are sent. Based on serum electrolyte results and physical findings, the attending endocrinologist recommends that IV fluids be started and hydrocortisone prescribed.

Newborn screening is a population-based public health program adopted in every state in the US as a health investment whose benefits far outweigh the costs. Every infant born in Rhode Island must be screened at birth for metabolic disorders and hemoglobinopathies in accordance with Rhode Island State law. These disorders can cause very serious problems for newborns if they are not detected and treated in a timely manner. Outcomes can include mental retardation, seizures, liver damage, emotional problems, hearing loss, overwhelming sepsis, and death. All of the disorders are rare.

At this time, newborns are screened for the following eight disorders: congenital adrenal hyperplasia, hypothyroidism, galactosemia, homocystinuria, maple syrup urine disease, phenylketonuria, biotinidase deficiency, and hemoglobinopathies (sickle cell disease). (Table 1).

Rhode Island has an integrated disease control program, assuring that screening, diagnostic and treatment services are provided for about 14,000 newborns each year. A key role of the Rhode Island Department of Health (RIDOH) Laboratory is case reporting, tracking and follow up for infants with abnormal results or unsatisfactory specimens. The RIDOH Laboratory follows all newborns to a point at which the screening status has been resolved and a treatment plan established for those newborns with positive test results. Last year we diagnosed 12 significant cases of genetic disorders. In addition, staff provided co-ordination or follow-up for approximately 20% of all specimens.

The program began in 1962 with screening for phenylketonuria (PKU). PKU is classified as an inborn error of metabolism in that the body is not able to metabolize the amino acid phenylalanine. If not detected and treated in the first few weeks of life, phenylalanine can accumulate to toxic levels and mental retardation will result. In 1976 laboratory testing was expanded and regionalized at the Massachusetts State Laboratory and the RIDOH Laboratory continued its role of coordinating all newborn screening functions.

Timing and communication are critical to the success of this program. Heel stick blood specimens are collected from newborns at 24-72 hours of age at each of the eight birthing hospitals in Rhode Island. These specimens are picked up at the hospitals on a daily basis by courier and transported to the RIDOH Laboratory. The specimens are then processed by RIDOH Laboratory staff and packaged for same day transport by courier to the Massachusetts State Laboratory for testing.

Abnormal results are phoned to the RIDOH Laboratory and providers are then immediately contacted with the information and arrangements for repeat testing are made. When no provider is identified, the RIDOH Laboratory staff is responsible for locating the family directly through a variety of means and assuring that the newborn is brought to medical attention. In most cases, the newborn will be referred at that time to a specialist or to a Department of Health-funded specialty clinic at Rhode Island Hospital for diagnosis, treatment and care coordination. Results on all other specimens are faxed to the RIDOH Laboratory for follow up and notification.

The RIDOH Laboratory is in the process of implementing a computerized tracking and reporting system (Neometrics Inc.) for newborn screening. At that time, all results and demographic information on newborns will be downloaded daily to the Neometrics computer from the Massachusetts Laboratory. The new computer system will greatly assist laboratory personnel in reporting results to the child's primary care provider, managing all follow up activities, tracking specimens to ensure that all newborns are tested, and providing the transmission of results (status) and demographics to the KIDS NET computer (a pediatric public health tracking system) for access by primary care providers. The partnership with KIDS NET will also improve outreach to families with no primary care provider.

In 1993, Rhode Island became the first state to implement universal screening for congenital hearing loss. The RI Hearing Assessment Program based at Women & Infants Hospital now manages the process of screening all infants born in any of the eight birthing hospitals in the state. Prior to hospital discharge, each baby undergoes a rapid, non-invasive screen for each ear. The program's audiologist interprets all results. For the small number who do not pass the initial screen, repeat screening and the program arranges follow up diagnostic evalua-

Table 1. Disorders being screened for; the year screening was begun.

PHENYLKETONURIA - 1962 * Severe mental retardation -evident by 6-12 months * Behavior problems * Hypopigmentation * Mousey odor of sweat and urine * Variant forms of PKU, known as hyperphenylalaninemia; the mental retardation is variable * Hyperphenylalanemia caused by defects of biopterin metabolism causes progressive neurological damage with seizures	**MAPLE SYRUP URINE DISEASE - 1965** * Poor feeding * Irritability * Vomiting * Central nervous sytem depression, coma, and then death
GALACTOSEMIA-1965 * Hypoglycemia * Liver damage * Jaundice * Failure to thrive * Lethargy * Sepsis, sometimes resulting in death	**HOMOCYSTINURIA-1965** * No signs in the newborn period * Later in life will develop the following: - seizures - osteoporosis - disocated lenses - liver damage - connective tissue damage - mental retardation - emotional problems
HYPOTHYROIDISM-1976 * Severe mental retardation * Poor muscle tone * Lethargy * Jaundice	**HEMOGLOBINOPATHIES-1990** * Sepsis in infancy * Jaundice * Anemia (microcytic hypochromic or hemolytic) * Splenic sequestration * Stroke
BIOTINIDASE DEFICIENCY-1995 * Seizures * Skin rash * Hair loss * Hypotonia * Ataxia * Hearing loss * Optic nerve atrophy * Developmental delay * Metabolic acidosis which xcan result in coma and death	**CONGENITAL ADRENAL HYPERPLASIA-1995** * Failure to thrive * Dehydration * Electrolyte abnormalities-death due to salt-losing crisis * Ambiguous genitalia * Excess adrenal androgen sequellae such as short stature and emotional disorders

tions. More details are available on the program's WEB site: http://www.health.state.ri.us/family/rihap.htm. The future of newborn screening has become a topic of regional and national significance over the past two to three years largely as a result of exploding new knowledge and the introduction of new testing technologies. Working in collaboration with other genetic service providers, Health Department representatives and consumers, the Newborn Screening Committee of the New England Regional Genetics Group recently issued guidelines for states to use in reviewing and making changes within their newborn screening programs. The Centers for Disease Control recently created a Public Health Genetics unit within its Center for Environmental Health and in collaboration with the Maternal and Child Health Bureau at the Health Resources and Services Administration has begun to develop national standards for newborn screening.

States differ in their willingness to mandate newborn screening. Community values and economic conditions will continue to predict as much as science and technology the future conditions to include in newborn screening programs. The RI Department of Health has formed an internal strategic planning task force to advise the Director of Health and the community on the future role of the Health Department in this new era of genetic technology. One immediate priority is to make sure that all stakeholders are offered an opportunity to be heard before changes are made to our state's Newborn Screening Program.

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Molecular Genetics for the Practitioner

Umadevi Tantravahi, PhD

The role of molecular genetics in medicine is expanding rapidly. The human genome consists of approximately 50,000-100,000 functional genes that encode a variety of proteins. Only 10% of the genome is made up of these genes (This category of the genome is called the "unique sequence" DNA). The remaining 90% of the genome is made up of various types of repetitive DNA and its function is less well understood. Individual genes themselves possess a structure that is less straightforward than had been assumed. The structure of most genes, for example, consists of coding regions called exons which represent the part of the gene which provides useful information for the production of proteins and non-coding regions called introns, whose function is not yet fully understood. All genes also contain regulatory sequences which provide directions to the cell as to if and when to produce a given protein, and how much of the protein to produce.

The entire complement of genetic material in a single cell is huge. It is therefore a significant feat of engineering to package a single molecule of human DNA which may measure up to a meter in length into the nucleus of a cell whose dimensions are measured in angstroms. Cells solve this topological problem by packaging DNA (along with proteins called histones) into highly ordered arrays, the most complex of which is the chromosome.

MOLECULAR GENETIC TESTING

The primary care physician or specialist will sometimes need to consider the genetic basis for a diagnosis. This requires the physician to be familiar with the available genetic tools, be able to order the appropriate test and be able to convey the results to the patient and family members in a meaningful way. Any one of these steps may require consultation with a clinical geneticist and/or a genetic counselor. The method of choice for diagnosis for a particular disease is based on what information is available for the disease to be tested.

Abbreviations Used:

ASO	allele specific oligonucleotide
CF	cystic fibrosis
DMD	Duchenne muscular dystrophy
DNA	deoxyribonucleic acid
FMR-1	Fragile X mental retardation syndrome
PCR	polymerase chain reaction
RFLPs	restriction fragment length polymorphisms

METHODS

Testing for single gene disorders

Most methods require DNA from the patient. In addition, based on the information being sought it may be necessary to obtain DNA from other family members also. DNA can be isolated from a variety of cell types such as peripheral blood, solid tissue, skin fibroblasts, dried blood spots, hair bulb and cheek cell scrapings. Some of the most frequently used techniques in genetic diagnosis are Southern blot

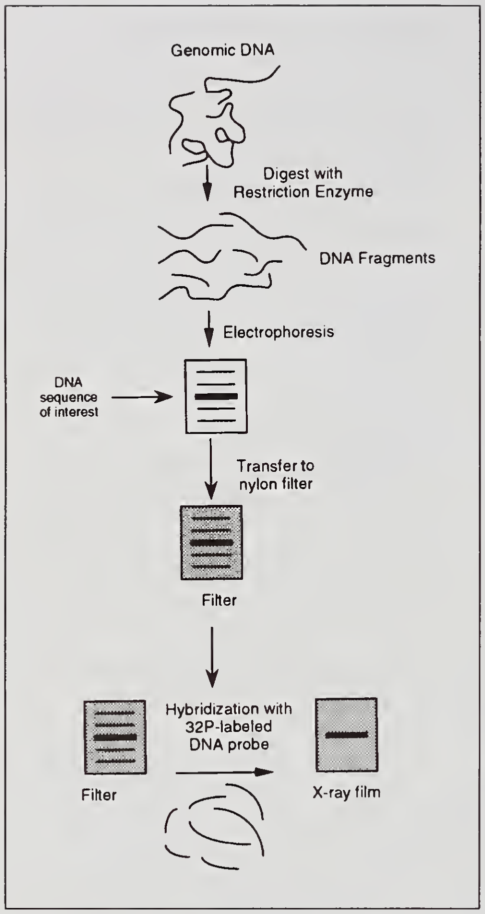
Genomic DNA
Digest with Restriction Enzyme
DNA Fragments
Electrophoresis
DNA sequence of interest
Transfer to nylon filter
Filter
Hybridization with 32P-labeled DNA probe
Filter
X-ray film

Figure 1. Southern blot analysis based on hybridization of probe DNA to target DNA and detection of a hybridization signal.

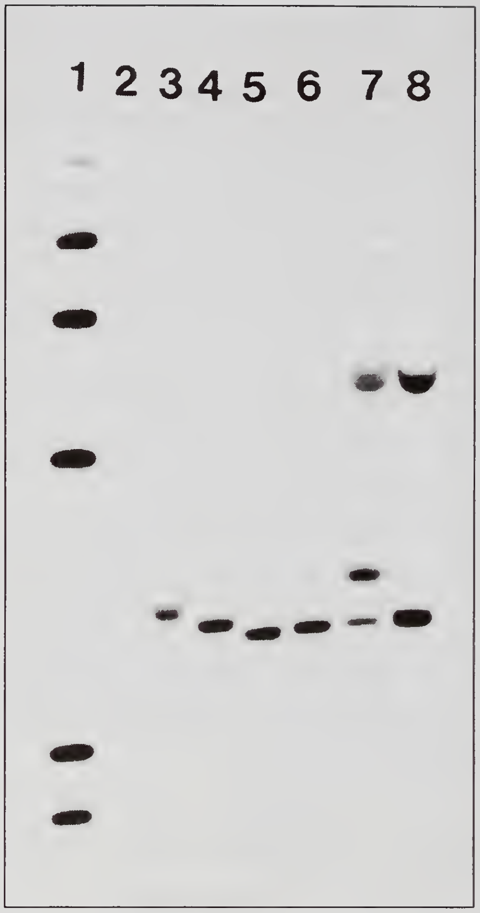

Figure 2. Southern blot analysis of Fragile X mutation. 1) size marker, 2) affected male, 3&4) pre mution males with different expansions of CGG repeats, 5) normal male, 6) affected female, 7) pre mution female, 8) normal female.

analysis for direct testing or linkage analysis, Polymerase Chain Reaction (PCR), Allele Specific Oligonucleotide (ASO) analysis for specific mutation detection and sequencing. There are other specialized tests that are used in some instances that are not routine and usually these are performed in the research laboratories.

Southern Blot and Linkage Analysis

This technique, developed by Dr. Southern in 1975, is based on hybridization of probe DNA to target DNA and detection of a hybridization signal (Figure 1). Cellular DNA is isolated from the patient and digested with restriction enzymes to generate an array of DNA fragments. Restriction enzymes are isolated from bacteria. These enzymes have the ability to recognize specific DNA sequences of four to six bases and cleave DNA at those sites. The fragments of DNA thus generated are size separated on agarose gels by electrophoresis. The DNA fragments in the gel are denatured (two strands of DNA are separated) and transferred to nylon membranes. These DNA fragments on nylon membranes are then hybridized to a specific DNA probe (for example, DNA fragments from a gene of interest). The DNA probe is labeled with a radioactive or non- radioactive tag. Probe signals are detected by autoradiography (radioactive) or enzymatic reaction (non-radioactive).

For example, in Fragile X mental retardation syndrome (FMR-1) the molecular defect is well characterized and can be diagnosed directly by using the gene probe and Southern analysis. FMR-1 is the most common cause of inherited mental retardation. It is an X linked recessive disorder with an incidence of 1/1200 males and 1/2500 females. The disease is caused by expansion of a trinucleotide repeat CGG within the FMR-1 gene. This triplet repeat varies in number from 6-54 in normal individuals. Female carriers and pre-mutation males have between 54-200 repeats. Affected individuals (full mutation) have 200 to 1000 repeats. Full mutation also confers other changes in the regulatory sequences. Southern blot analysis using part of the gene that contains the repeat sequence can be used to determine the mutation status directly. Because of the increase in the number of repeats in pre-mutation and full mutation individuals, the gene probe in Southern blot analysis will detect a larger size fragment compared to normal controls (Figure 2).

However, for many genetic diseases, the exact molecular basis is not known. In these situations, diagnosis can be made by indirect testing called

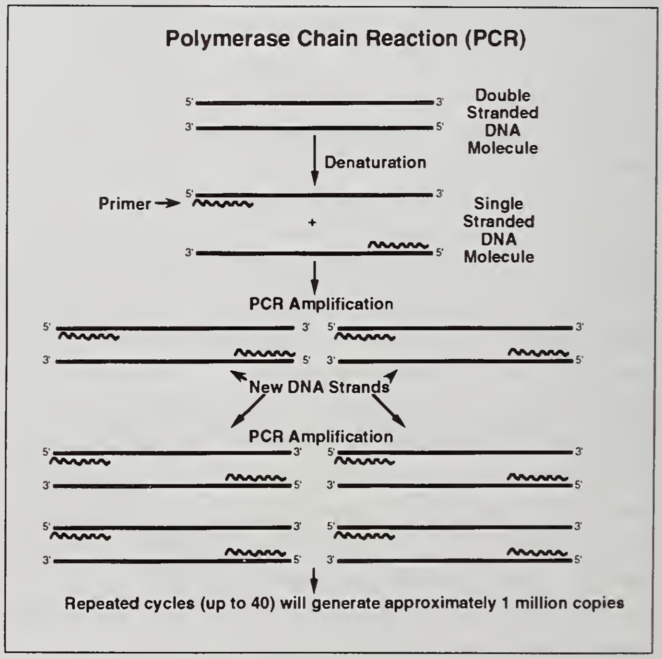
Polymerase Chain Reaction (PCR)
5'
3'
3'
5'
Double Stranded DNA Molecule
Denaturation
Primer
Single Stranded DNA Molecule
PCR Amplification
New DNA Strands
PCR Amplification
Repeated cycles (up to 40) will generate approximately 1 million copies

Figure 3. Polymerase Chain Reaction (PCR). By repeat heat denaturation, annealing and polymerization, millions of copies of the same DNA sequence can be generated.

linkage analysis. Linkage analysis is based on following the inheritance of known DNA markers that are closely linked to the gene or within the gene itself. To be informative, differences in DNA sequences known as polymorphisms must exist between the two alleles at a locus. When DNA is digested with restriction enzymes, because of the subtle differences in the DNA sequences, different sizes of fragments (Restriction Fragment Length Polymorphisms (RFLPs)) are produced that are detected by the DNA probe of interest using Southern blot analysis. For linkage analysis, DNA samples from parents of the patient and other family members are analyzed to determine the carrier status and diagnosis. Before the genes for diseases such as sickle cell disease, cystic fibrosis, Huntington's disease and Duchenne muscular dystrophy were isolated and mutations were characterized, linkage analysis with RFLP DNA markers was used for diagnosis.

The Polymerase Chain Reaction (PCR)

Polymerase Chain reaction technique is the most powerful, yet conceptually simple, technique for the analysis of DNA and RNA. Developed by Dr. Sakai in 1985, PCR can be used to generate millions of identical copies of a single DNA molecule in a few hours without having to go through the techniques of cloning. This technique has revolutionalized the field of molecular biology especially molecular diagnostics. PCR is based on the enzymatic amplification of a DNA fragment using small synthetic complementary DNA fragments called "primers." A very small amount of genomic DNA is mixed with the synthetic DNA primers that flank the region of a gene to be amplified along with deoxy ribonucleotides and the enzyme DNA polymerase. Under optimum conditions, the primers attach to the homologous region in the DNA and using the nucleotides, new complimentary DNA strands are synthesized by the DNA polymerase. By repeated heat denaturation, annealing and polymerization, millions of copies of the same DNA sequence are generated (Figure 3).

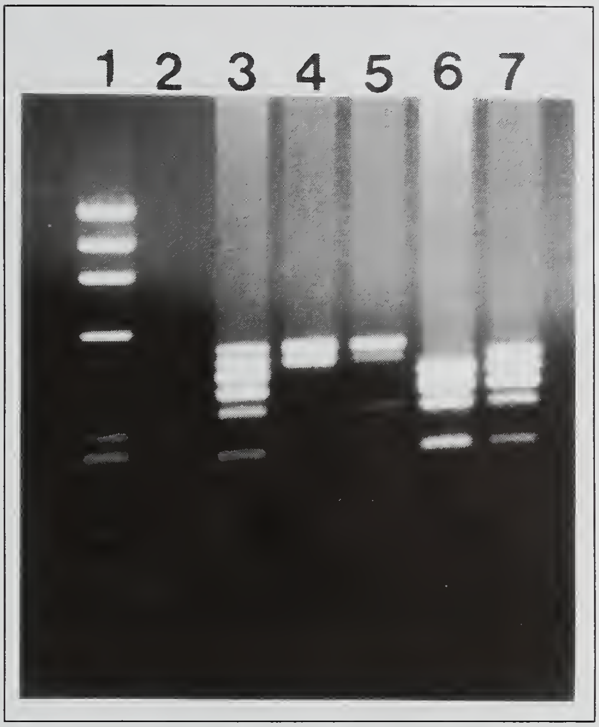

Figure 4. Multiplex PCR analysis of DMD gene. 1) size marker, 2) no DNA control, 3) normal male, 4-7), examples of deletions of different exons.

The amplified DNA can be analyzed using various techniques based on the mutations being studied. For example, in Duchenne muscular dystrophy (DMD), an X linked recessive neuromuscular disorder, 70% of the gene defects are due to deletions of DNA sequences in the gene. These deletions are heterogeneous among patients. The gene is cloned and the sequence of most of the exons (regions that encode proteins) is known. Based on the sequence information, PCR primers to detect 98% of the mutations due to deletions are designed. Using a mixture of these primers, patients' DNA is amplified and analyzed for deletions. The amplified products are separated on agarose gels and visualized by fluoroscopy. If there is a deletion, the primer specific for that region of DNA will not amplify and no signal will be seen on the gels (Figure 4). Another 30% of the mutations are analyzed using linkage analysis as described previously.

The primary care physician or specialist will sometimes need to consider the genetic basis for a diagnosis. This requires the physician to be familiar with the available genetic tools, be able to order the appropriate test and be able to convey the results to the patient and family members in a meaningful way.

In cystic fibrosis (CF), a different technique is used to analyze the PCR products. CF is the most common autosomal recessive disease in North American Caucasians of European ancestry. Mutation in the CF transmembrane conductance regulator results in CF. The most common mutation (75%) is due to a deletion of three base pairs which results in the loss of amino acid phenylalanine at position 508. However, more than 400 different mutations are documented and some are specific for certain ethnic groups. DNA to be tested is amplified with primers flanking the region of mutation. The amplified DNA is spotted onto nylon membrane using suitable apparatus in duplicate. Two short synthetic oligonucleotide probes (ASO), one complimentary to the normal allele and the other for the mutated allele are synthesized. The ASO is labeled with a radioactive molecule and hybridized to the nylon membrane containing the amplified DNA to be tested. If a mutation is present in the test DNA, only the ASO for the mutant allele will give a positive signal. Using this technique, several mutations can be screened in a relatively short amount of time.

There are several other techniques such as DNA sequencing, denaturing gradient gel electrophoresis and single strand conformation polymorphisms that are used to detect known and unknown mutations using PCR amplified DNA.

CONCLUSIONS

With the tremendous advances in the recombinant DNA technology and the Human Genome Initiative, exciting and rapid progress is being made in the field of Human Molecular Genetics and disease gene identification. It is the responsibility of the physician to be aware of these developments and incorporate them into practice. A good starting point is to maintain a working relationship with the genetic professionals (clinical geneticists, genetic counselors and laboratories providing diagnostic tests). There are several Web sites for genetics information including sites for specific diseases. In future, advances in the field of molecular genetics and our ability to diagnose disease at molecular level hopefully will lead to efficient treatment strategies for genetic disorders.

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Conventional and Molecular Cytogenetics in Medicine

Hon Fong L. Mark, PhD, FACMG

Clinical cytogenetics is a relatively new specialty of medical genetics. Historically modern genetics began in 1900 with the rediscovery of Gregor Johann Mendel's laws of inheritance based on his experiments with the garden pea.

In 1914, Theodor Boveri proposed the somatic mutation theory of cancer, which first suggested the clonal origin of neoplasms. Boveri proposed that cancer developed from a single cell that acquired a genetic alteration. However, Boveri's hypothesis could not be tested with the technology available at that time. With the advent of techniques for analyzing chromosomes, support of Boveri's hypothesis began to appear in the literature in the 1960s. Today cancer genetics is one of the most exciting areas of medical genetics.

Analysis of chromosomes was technically difficult until the serendipitous discovery of a hypotonic solution for spreading chromosomes, the use of colchicine to arrest metaphases, and the development of an optimized protocol for lymphocyte culture in the presence of phytohemagglutinin (PHA).[1,2] In 1956, the correct number of chromosomes in man was established as 46, not 48.[3]

Abbreviations Used:

ABMG	American Board of Medical Genetics
ABMS	American Board of Medical Specialties
ACMG	American College of Medical Genetics
ASHG	American Society of Human Genetics
BMT	bone-marrow transplants
CML	chronic myelogenous leukemia
CMSS	Council of Medical Specialty Societies
FISH	fluorescent in situ hybridization
PHA	phytohemagglutinin

CONVENTIONAL CYTOGENETICS IN MEDICINE

In 1960, Nowell and Hungerford reported the first consistent chromosomal abnormality associated with a single cancer phenotype, chronic myelogenous leukemia (CML).[4] The marker chromosome was named the Philadelphia chromosome (Ph) in honor of the city where it was discovered. This constituted one of the earliest pieces of evidence supporting the chromosome theory of cancer. Unfortunately, few other consistent chromosomal changes could be discerned until the advent of various banding techniques in the 1970s, allowing many more specific chromosomal rearrangements to be identified. For example, the Philadelphia chromosome was not revealed to be a reciprocal translocation between chromosomes 9 and 22 until 1973.[5]

In 1980 the American Board of Medical Genetics (ABMG) was formed under the sponsorship of the American Society of Human Genetics

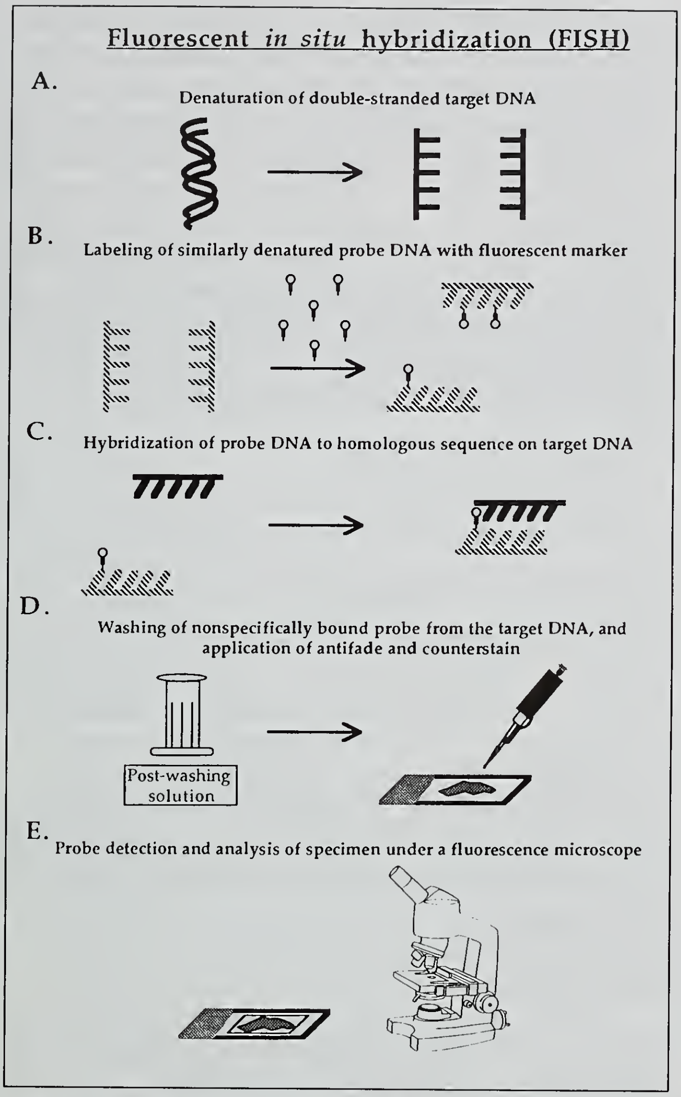
Fluorescent *in situ* hybridization (FISH)
A.
Denaturation of double-stranded target DNA
B.
Labeling of similarly denatured probe DNA with fluorescent marker
C.
Hybridization of probe DNA to homologous sequence on target DNA
D.
Washing of nonspecifically bound probe from the target DNA, and application of antifade and counterstain
Post-washing solution
E.
Probe detection and analysis of specimen under a fluorescence microscope

Figure 1. The basic steps of FISH. Reprinted with permission from *Cytobios* 1997; 90: 7-22.

(ASHG). Commercial cytogenetics laboratories began to appear and flourish alongside academic centers. Also during the 1980s the polymerase chain reaction was developed. Newly developed molecular diagnostic techniques was applied to cytogenetics, from the creation of DNA probes for identifying and mapping oncogenes, to the widespread use of fluorescent probes for chromosome staining as an adjunct to banding.

MOLECULAR CYTOGENETICS IN MEDICINE

Although conventional cytogenetics via various banding techniques is a highly informative and established diagnostic procedure which permits the inspection of the entire human genome at a glance, its application is restricted to dividing cells. Interpretation is also difficult with an inadequate number of high-quality metaphases. The advent of fluorescent in situ hybridization (FISH), made possible through recombinant DNA technology, offers an unprecedented opportunity for the analysis of large numbers of non-dividing cells in interphase as well as metaphase. Thus, FISH is sometimes called interphase cytogenetics. The basic steps of FISH are summarized in Figure 1. Examples are given in Figure 2. In the remaining space I shall provide glimpses of the excitement of the field through selected cases from the archives of the Rhode Island Hospital Cytogenetics Laboratory, founded in 1963 as one of the first in the country.

FISH FOR MARKER CHROMOSOME IDENTIFICATION

During routine cytogenetic analysis, an unidentifiable chromosome called a marker is occasionally encountered. An International System for Human Cytogenetic Nomenclature[6] defines a marker chromosome as a structurally abnormal chromosome in which no part can be identified.

A case report by Abuelo, Mark and Bier[7] described how FISH was used to identify the origin of a marker in a child with developmental delay. The marker was demonstrated to be an inv dup (15), or pseudo-dicentric.[6] As in other reported cases, conventional techniques could not determine the origin of the extra chromosomes. Only with recent advances in molecular cytogenetic technology could many supernumerary chromosomes be identifiable.[7]

MICRODELETION DETECTION USING FISH AND LOCUS-SPECIFIC PROBES

A microdeletion is a small deletion of chromosomal material. FISH is one of the most powerful methods for detecting such structural rearrangements. Some of the specific applications of FISH include the detection of deletions and cryptic translocations in Miller-Dieker Syndrome (17p13.3), and deletions in Prader-Willi and Angelman Syndromes (15q11.2), deletions in DiGeorge and Velocardiofacial Syndromes (22q11.2). For many microdeletion syndromes, a combination of routine cytogenetics and FISH analysis may be appropriate. Included in this list of syndromes are 17p11.2 deletions in the Smith-Magenis Syndrome, 7q11.23 deletions in the Williams Syndrome, 4p16.3 deletions in the Wolf-Hirschhorn Syndrome, 5p15.2 deletions in the Cri-du-Chat Syndrome, and Xp22.3 deletions in Steroid Sulfatase Deficiency/Kallman Syndrome.[8]

Molecular cytogenetics can also detect microduplications such as the microduplications at 17p11.2 which leads to Charcot-Marie-Tooth disease (Type 1A).[8]

SEX CHROMOSOME IDENTIFICATION AND DETECTION OF MOSAICISM

The utility of FISH in detecting mosaicism was illustrated by a case report by Mark et al.[9] The patient was an 18-year-old woman referred for delayed puberty, short stature, hypergonadotropic hypogonadism, primary amenorrhea, and growth retardation. Cytogenetic analysis was ordered to rule out a chromosomal basis for her clinical findings. GTG-banding analysis of peripheral blood lymphocytes revealed the presence of predominantly 46,XY cells. Using dual-color X and Y chromosome-specific probes, it was found that 87% of her cells were 46,XY and 9% of her cells were 45,X. To establish unequivocally the presence of mosaicism, a skin biopsy was obtained for fibroblast culture, which further corroborated the results of the periph-

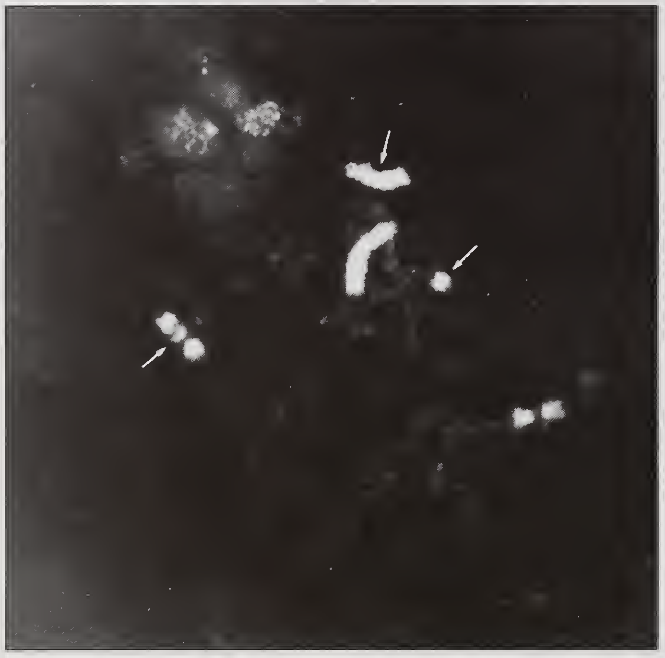

Figure 2. Fluorescent in situ hybridization illustrating the use of chromosome painting probes for the detection of translocations. Reprinted from *Medicine & Health/Rhode Island* 1996; 79: 333-335.

eral blood study. The unequivocal presence of XY cells put the patient at risk for neoplastic transformation of the gonads. Laparoscopy and surgical removal of the patient's presumptive streak gonads were therefore undertaken. Follow-up results on the gonadal tissues confirmed the previous findings.

In another study, a combined cytogenetic and molecular approach was employed to detect low-percentage mosaicism for a minor cell line in a fifteen-year-old patient with delayed puberty and short stature.[10] The use of DNA analysis further helped delineate the nature of a marker present in a low percentage of cells initially detected using cytogenetics.

FISH FOR CANCER STUDIES

One of the most important uses of FISH is for chromosome enumeration in the detection of aneuploidies such as trisomy 8 and monosomy. Mark et al,[11] for example, used FISH to assess the proportion of cells with trisomy 4 in a patient with acute non-lymphoblastic leukemia, and recognized FISH as a potentially useful tool for monitoring minimal residual disease. Another study described congenital trisomy 8 mosaicism in gestational trophoblastic disease.[12] Subsequently, archival materials from this and other patients were retrieved, and FISH was used to assess chromosome 8 copy number.[13] Trisomy 8 in cancer has been studied extensively in this laboratory.[14]

One of the most important applications of FISH in cancer is the monitoring of engraftment in sex-mismatched bone-marrow transplants (BMT). By detecting the ratio of XX to XY cells using FISH and the dual color X and Y chromosome probes on a post-BMT sample, the success of engraftment as well as the risk of recurrence can be assessed. Translocation probes, such as bcr/abl and t(15;17), can also be used to monitor disease status. For example, our laboratory has identified a unique complex translocation involving chromosomes 9, 13, 15, 17 and 22 associated with Ph chromosome-positive CML using such an approach.[15]

FISH can also be used to characterize cell lines derived from primary tumors. Cervical cancer cells with multiple numerical and structural chromosomal abnormalities are described by Mark et al.[16] Oncogene amplification can be detected in archival materials by FISH, which has proven to be superior to the traditional method of immunohistochemistry. HER-2/neu amplification has been the subject of several on-going research studies in this laboratory.[17]

INTERPHASE CYTOGENETICS

Conventional cytogenetics has been hampered by the requirement that there be sufficient dividing cells with adequate quality for karyotypic analysis. FISH is useful when other sources for conventional cytogenetic analysis, such as peripheral blood or bone marrow, are not available.[18] Buccal smears, a noninvasive technique, can be utilized in the absence of a more suitable specimen, and can supplement the pool of scorable cells to detect low-percentage mosaicism.

Similarly, FISH can be performed on cervical and semen smears, frozen or fixed suspensions of blood, bone marrow, and tumor cells. Hair follicles, urine sediments, and other exfoliated non-dividing cells are also amenable to FISH analysis.

CONCLUSION

With the arrival of the 1990s came the recognition of the American Board of Medical Genetics (ABMG) as the 24th primary specialty board of the American Board of Medical Specialties (ABMS). The American College of Medical Genetics (ACMG), founded in 1991, is the clinical standards and practice arm of the medical genetics community. In 1995 the ACMG became a full member of the Council of Medical Specialty Societies (CMSS). It received a seat in the American Medical Association's House of Delegates in 1996.

With often newsworthy advances in human genetics, medical geneticists are increasingly brought under close scrutiny by their peers, the public, medical regulators, and legislators. Currently, clinical laboratories in the United States are certified under the Clinical Laboratory Improvement Amendments of 1988 (CLIA 88) and cytogenetics laboratories must be registered with the Health Care Financing Administration (HCFA). The ACMG has also assumed responsibility for developing standards of practice for the medical genetics community.[19] In addition, most, if not all, cytogenetics laboratories are inspected by the College of American Pathologists' Laboratory Accreditation Program and participate in the Inter-laboratory Comparison (Surveys) Program. Issues related to setting standards in the clinical cytogenetics laboratory have been discussed elsewhere.[20,21,22]

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Cystic Fibrosis

Mary Ann Passero, MD

Abbreviations Used:

ATP	adenosine triphosphate
CBAVD	congenital bilateral absence of the vas deferens
CF	cystic fibrosis
CFTR	CF transmembrane regulator
PEP	positive expiratory pressure

Cystic fibrosis (CF) is one of the most common life-shortening recessive genetic disorders. Its phenotypic features include chronic bacterial infections of the sinuses and lungs, obstructive lung disease with bronchiectasis, exocrine pancreatic insufficiency, excessive sweat salt loss and male infertility.[1] Approximately 21,000 United States residents are currently diagnosed with CF; about 100 live in Rhode island.[2] In 1989 the gene responsible was identified on the long arm of chromosome 7 and its nucleic acid and amino acid sequences were identified.[3] This gene encodes a cyclic adenosine monophosphate regulated chloride channel called the CF transmembrane regulator (CFTR).[4] CFTR is generally located on the apical surfaces of epithelial cells. CFTR not only functions as a chloride channel itself but also channels adenosine triphosphate (ATP) to the epithelial surface to activate other chloride channels. It may also directly or indirectly inhibit sodium channel function. Mutations in the CFTR gene result in defective chloride channel transport in the epithelial cells of the respiratory, gastrointestinal and reproductive tracts and in hepatobiliary, sweat and pancreatic ducts.[5] Defective CFTR in the reabsorptive duct of the sweat gland accounts for the elevated chloride levels found in CF sweat. It is less clear how alterations in CFTR function lead to sinopulmonary infections, neonatal intestinal obstruction (meconium ileus), hepatobiliary disease, pancreatic insufficiency, etc.

More than 700 different mutations of CFTR have been identified. The delta F508 mutation (the deletion of phenylalanine at amino acid number 508 in the CFTR sequence) is the most common. Of the over 12,000 CF patients in the United States who have been genotyped, almost 50% are homozygous for delta F508 and another 35% have one delta F508 mutation.[2] Some CF mutations are associated with pancreatic insufficiency at birth or soon after, while others are associated with pancreatic sufficiency or late onset pancreatic insufficiency. When one patient has two different CF mutations (for example, delta F508 and R117H), his phenotype will correlate with the "milder" (R117H) mutation, and he will be pancreatic sufficient. In general, mutations which produce more functional CFTR products are associated with milder clinical courses. However, much variability in clinical presentation and progression of illness exists even among siblings with apparently identical mutations.[6]

The diagnosis of CF is made on clinical grounds. It requires a sweat chloride (obtained by quantitative pilocarpine iontophoresis) of greater than 60 meq./l and at least one of the following: a) chronic suppurative obstructive lung disease, or b) pancreatic insufficiency, or c) a sibling or first cousin with CF. Another combination considered diagnostic is a positive sweat chloride and obstructive azoospermia (congenital bilateral absence of the vas deferens).[7]

Although genotyping is helpful in confirming the diagnosis of CF or determining carrier states, it is not as diagnostically useful as the sweat test. Only 70% of recognized CF patients have two identifiable mutations, and some individuals with two identifiable mutant CFTR genes may have an ameliorating or neutralizing second mutation elsewhere.[6]

In patients with equivocal sweat test results, late onset lung disease and pancreatic sufficiency, or equivocal or high sweat chloride results and only suggestive clinical findings such as pancreatitis or nasal polyps, the diagnosis may be difficult or impossible. Stern has suggested the following guidelines for the "rational use of clinical signs and laboratory tests to diagnose or rule out CF."[8]

A negative sweat test (presuming all guidelines for a good sweat test are met[9]) can rule out CF for:

1) patients with symptoms of exocrine pancreatic insufficiency (e.g., malabsorption, steatorrhea) or a complication strongly linked to pancreatic insufficiency such as hypoproteinemia, anasarca of infancy, rectal prolapse in childhood, distal intestinal obstruction syndrome or hepatitic cirrhosis before age 30. His rationale is that for the pancreas to be affected, CFTR function must be well below the level for normal sweat gland function.
2) infants who present with hyponatremia and heat prostration
3) patients with a family history of "classic" CF
4) children being considered for adoption
5) patients with pulmonary symptoms such as recurrent pneumonia or persistent cough but otherwise not consistent with CF

Although involvement of the pancreas in CF requires functional CFTR levels lower than that required for abnormal sweat chloride, respiratory and sweat gland diseases may appear at higher levels of functioning CFTR. Thus, a normal or equivocal sweat chloride in a child or adult with respiratory symptoms highly suggestive of CF, such as nasal polyps, pansinusitis or chronic suppurative lung diseases (especially if *P. aeruginosa* grows from respiratory secretions or if there is digital clubbing) cannot rule out the diagnosis. In such patients, genotyping should be done. If the genotyping is inconclusive, semen analysis, sinus x-rays for pansinusitis or bronchial lavage looking for *Pseudomonas* may be helpful. An increased nasal potential difference across the airway is another diagnostic tool.[9] Potential difference studies for CF are done well in few places in the United States.[10] *Sometimes a patient with an inconclusive diagnosis must simply be followed and treated empirically for several years until the clinical course proves or disproves the diagnosis.*

Similarly, when pancreatitis or cholelithiasis in patients under 30 years of age cannot be explained by another diagnosis, the sweat test is negative or equivocal, and CF genotyping is inconclusive, then sinus x-rays, semen analysis, and potential difference across the airway may be helpful. One may even consider bronchoalveolar lavage, looking for evidence of suppurative bronchitis in these patients with no pulmonary symptoms. Again, sometimes we may need to observe the patient for several years before making the diagnosis.

Congenital bilateral absence of the vas deferens (CBAVD) as the presenting sign for CF warrants special consideration. CBAVD is almost universal in "classic" CF. Embryologic development of the vas deferens apparently requires more functional CFTR than is required for normal functioning of other organs affected by CFTR mutations.

It is not rare to have CBAVD, normal or near normal sweat electrolytes and one or even two identifiable CFTR mutations and no other clinical manifestations of CF. A mutation of intron 8 of the CFTR genes is associated with CBAVD. This intron normally contains a sequence of 7 or 8 thymidines (7T or 9T). The number determines the efficiency of expression of exon 9 of CFTR. If intron 8 has only 5 thymidines (5T) and the patient is either homozygous for 5T or has another more severe CFTR mutation in addition to 5T, then not enough functioning CFTR will be produced for normal development of the vas deferens. Genetic counseling is required for CBAVD if *in vitro* fertilization by means of sperm extraction is to be considered. The patient's wife may be an unsuspecting carrier of a CF mutation.[6]

> *The diagnosis of CF is made on clinical grounds. It requires a sweat chloride (obtained by quantitative pilocarpine iontophoresis) of greater than 60 meq./l and at least one of the following: a) chronic suppurative obstructive lung disease, or b) pancreatic insufficiency, or c) a sibling or first cousin with CF.*

◆

CF may be diagnosed by chorionic villus sampling in the first trimester of pregnancy or amniocentesis in the 2nd trimester if the parents are known carriers; e.g., they have already had a child with CF. Using DNA polymorphisms as markers, diagnostic accuracy is nearly 100% even when the patient's particular mutations are not identifiable with DNA probes. Using polymerase chain reactions to amplify DNA from a single cell, it is even possible to diagnose CF in eggs fertilized in vitro before implantation.[11]

Carrier state in CF family members can be detected with great accuracy if the mutations are known but CF carrier state screening in the general population is problematic. About 10% of CF mutations will not be detected using currently available probes.[12]

Neonatal screening for CF is available in some states assaying trypsinogen from dried blood samples. Positive samples are screened using the most common CF DNA probes. If both tests are positive, the infant's family is notified and a sweat test is performed. Neonatal screening remains controversial because at least 15% of CF infants would go undetected. Even with the two-step approach there are still a number of false positive results. Early detection, however, provides an opportunity to prevent neonatal malnutrition in certain infants with CF. It is not universally accepted that the current demonstrable benefits of neonatal screening outweigh the risks and justify the costs.[13]

Most CF morbidity is related to fat and protein malabsorption and progressive suppurative obstructive lung disease. However, many patients develop significant sinus disease, liver disease, neonatal or recurrent episodes of distal intestinal obstruction, recurrent heat prostration, bone and joint diseases as well as psychosocial problems related to having a chronic progressive and life-shortening disease.

The development of a national network of CF centers, with an expert team of physicians, surgeons, nurses, nutritionists, respiratory therapists, social workers, and genetic counselors, has contributed to the median life expectancy and quality of life for CF patients.[14]

Malabsorption in CF is primarily treated with oral pancreatic enzyme replacement at meals and snacks. Enteric coated pancreatic products and vitamin supplements can control malabsorption in 85% of patients. Some patients may also require bicarbonate replacement, bile salt replacement, or the addition of H2 blockers to optimize pancreatic enzyme replacement function. Even patients who are optimally treated may still lose 10 to 20% of ingested calories.[14]

Neonates who have required surgery for meconium ileus with atresia or who have concurrent significant respiratory disease may require predigested formulas and/or nasogastic or gastrostomy feedings. Gastroesophageal reflux may complicate the feeding problems of CF infants and older CF patients with chronic cough. Medical management with gastrointestinal motility agents and/or H2 blockers or proton pump inhibitors may be necessary. Surgical fundal plication may also be helpful in selected patients.

CF patients with advanced lung disease require intense nutritional attention. The addition of oral caloric supplements to their usual high calorie diet may be helpful if the lung disease is only moderate, as may behavior modification counseling to increase caloric intake.[15] Eventually poor appetite and the high caloric demands of chronic infection, cough and copious mucopurulent secretions will make it impossible to eat enough to maintain good nutrition and growth, especially true during adolescence. Nocturnal gastrostomy feedings and even parenteral nutrition through a central line may be necessary to maintain ideal body weight. Not only is adequate nutrition necessary to optimize the patient's immune status but optimal results for lung transplantation require maintaining at least 80% of ideal body weight to the time of surgery.[15]

Progressive obstructive pulmonary disease is the primary cause of CF morbidity: 90% of CF deaths are from respiratory failure. Endobronchial infections have been detected in early infancy. Initally *Staphylococcus aureus*, *Hemophilus influenzae* and other gram negative bacteria colonize the airway. By age 10 *Pseudomonas aeroginosa* gradually becomes the prominent pathogen in most patients.[16] An intense inflammatory response with massive infiltration of the airways by neutrophils is characteristic.[17]

Current treatment of CF lung disease is empiric. The consensus *Clinical Practice Guidelines for Cystic Fibrosis*, published by the Cystic Fibrosis Foundation in 1997, describes an aggressive program of comprehensive routine monitoring and early intervention to promote mucociliary clearance, reduce the burden of bacterial infection and control airway inflammation.[18] Such intervention practiced in CF centers throughout the United States has contributed to the gradual increase in median survival from 14 years in 1969 to 31 years in 1997.[2]

In general, monitoring is done during clinic visits at 3-month intervals. An interval history, a physical examination and culture of sputum or deep nasopharyngeal secretions are done. When the patient is old enough to cooperate, pulmonary function testing is done every 6 months. FEV1 remains the best indication of progression of CF lung disease.[19] Serial chest x-rays every one to two years and with exacerbations of respiratory symptoms are also helpful.

Techniques to enhance mucociliary clearance are usually prescribed for all patients. Postural drainage and chest percussion of all lung lobes once or twice a day has been the traditional prescription. These time-consuming techniques disrupt normal family lifestyle; and adherence is generally low. Alternative mucus clearance methods include: active cycle breathing, positive expiratory pressure (PEP) devices, autogenic drainage, the therapy vest, the Flutter device, and the intrapulmonary precussive device.[20-23] Exercise programs have also been developed to enhance mucociliary clearance. No long-term studies have compared efficacy or likelihood of compliance among these various regimens. Most care providers offer patients 2 or 3 alternatives. Many patients switch from one technique to another depending on their age, need for independence, or progression of their lung disease.

The abnormal viscosity of CF sputum is primarily the result of extracellular DNA released from neutrophils. Recombinant human DNase (dornase-alpha) has replaced N-acetyl cysteine as the preferred mucolytic agent for CF. Dornase by aerosol once a day added to routine therapy in stable CF patients increases FEV1 by 5.6% and reduces the age-adjusted risk of a pulmonary exacerbation by 28%. Clinically it also reduces daily cough in mild to moderate lung disease. It is not clear whether the regular use of dornase will actually delay the progression of CF lung disease.[24]

Most CF patients demonstrate bronchial hyperactivity at least some of the time and most patients will demonstrate improved pulmonary function after inhaled albulterol. Metered dose or aerosolized beta adrenergic agents are commonly used prior to chest PT. Periodic monitoring with PFTs before and after bronchodilator will identify the occasional patient who has a paradoxical response.[25]

The upper and lower airways of CF patients become colonized with pathogens early in life. Long-term therapy with oral and inhaled antibiotics has been used for many years with the hope of reducing the frequency of hospitalizations and slowing the progression of pulmonary obstruction.[16-26] Inhaled tobromycin nebulized as a 300mg. per 5ml. formulation twice a day in cycles of 4 weeks on and 4 weeks off has been shown to reduce sputum bacterial density, decrease the need for hospitalization and intravenous antibiotics and moderately increase pulmonary function in CF patients colonized with susceptible *P. aeruginosa*.[27] Pulmonary exacerbations usually require hospitalization for parenteral antibiotics and a vigorous program of chest physiotherapy.

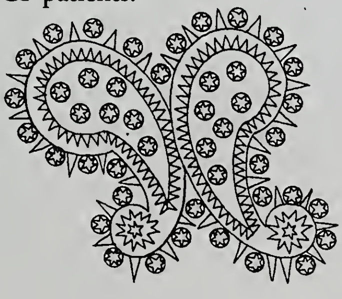
Antibiotic therapy is directed by sputum culture and antibiotic sensitivity studies. Treatment for 10 to 21 days is usually required to return the patient to his baseline. At least two antibiotics are usually used to cover *P. aeruginosa*, a beta lactam or quinolone and an aminoglycoside.

Because of the exuberant airway inflammation in CF, a variety of anti-inflammatory agents have been tried. CF patients treated with alternate day steroids at 1 mg/kg/dose show improved pulmonary function compared to placebo, but develop significant growth regardation. Inhaled beclomethasone at 400 micrograms per day does not show measurable improvement in inflammatory markers, but higher dosages of more potent steroids have yet to be adequately evaluated.[17]

High-dose ibuprofen (20-30mg/kg up to 1600 mg b.i.d. to achieve plasma levels of 50 to 100 micrograms per ml) has been shown to slow the progression of pulmonary disease in mildly affected patients (FEV1 greater than 80% of predicted). In 5 to 13 year-olds, the average rate of decline of FEV1 was reduced by 88% compared to placebo. Older patients did not show so dramatic an effect. A pharmaceutical study to optimize plasma levels is necessary before therapy is initiated. Low plasma levels of ibuprofen actually increase the inflammatory response in the airway.

In the past 10 years, double lung transplantation has become an option for many CF patients with end-stage lung disease. About 60% of CF patients who receive lung transplants survive more than 5 years. Immunosuppression and the threat of infection and rejection remain lifelong concerns. Successful double-lung transplantation, however, promises a more normal lifestyle without dyspnea. Patients should be referred for lung transplantation when death from CF lung disease is highly likely within 3 years. The current waiting time for cadaver lungs is 18 to 24 months. Infection and technical complications such as primary graft rejection, bleeding and bronchial dehiscence account for most early deaths. Chronic rejection with the development of bronchiolitis obliterans and infection account for most later deaths.[15]

Clearly, there is a need for more definitive therapy for CF. Various methods aimed at replacing the defective gene or enhancing the efficacy of the mutant CFTR are under investigation. Until there is a cure, we must make optimum use of current diagnostic and therapeutic tools to prolong survival and enhance the quality of life for CF patients.



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Presymptomatic Testing for Huntington's Disease: The Role Of Genetic Counseling

Amanda Walsh, MS

Genetic counseling is a crucial component of presymptomatic testing for Huntington's disease (HD). It includes a discussion of the clinical aspects of HD, the molecular genetics and inheritance of HD, motivations for testing, possible ramifications of testing as well as assessment of an individual's support system and psychological health. The role of genetic counseling in presymptomatic testing for HD is well recognized. In 1994, The Huntington's Disease Society of America guidelines recommended pretest genetic counseling, psychological screening, and neurological evaluation.[1] Subsequently, many laboratories began to require counseling prior to presymptomatic testing. In our center counseling involves four separate visits, the third of which is a full neurological evaulation. This allows an individual time to assimilate information and make a thoughtful decision regarding testing. This article will review the process of genetic counseling for presymptomatic HD testing.

CLINICAL ASPECTS

Genetic counseling begins with a review of the clinical aspects of HD, a degenerative neurological disorder which includes motor, cognitive, and emotional disturbances. Motor abnormalities include both involuntary and abnormal voluntary movements. Progression of motor abnormalities results in severe rigidity and joint contractures. Cognitive losses are progressive and typically begin with a decrease in cognitive speed and flexibility as well as impaired concentration. Psychiatric disturbances in HD are common. Typical symptoms include depression, irritability and apathy.[2] Although some families are knowledgeable about the symptoms, others are less so. This may be due to a lack of involvement with affected family members, attributing symptoms to individual personalities, or a negative family history of HD. Individuals contemplating presymptomatic genetic testing should understand the symptoms and progression of HD, so that they can determine how this disease may impact their lifestyles, goals and relationships.

At this time no cure exists, although symptomatic treatment is available. Both high potency neuroleptics and Dopamine depleting agents have been used to reduce chorea.[2] The psychiatric symptoms associated with HD have been treated with tricyclic antidepressants, selective serotonin reuptake inhibitors and monoamine oxidase inhibitors.[2] Unfortunately, no treatment has been proven to reduce cognitive abnormalities.[2] Prior to presymptomatic testing, individuals should understand that symptomatic treatment exists, but none of these treatments ameliorate the disease.

INHERITANCE

During genetic counseling, it is explained that HD is inherited in an autosomal dominant fashion; that is, each offspring of an affected individual is at 50% risk for the condition. For counseling purposes, the risk reduces with age when an individual is asymptomatic. Therefore, a 25 year old and a 55 year old, both with an affected parent, have an age adjusted risk of 49% and 25% respectively.[7] Individuals undergoing presymptomatic testing for HD must understand their risk for the condition. Additionally, if they are affected, it is possible that their age at onset will be earlier than in their parents.

MOLECULAR GENETICS

A discussion of the molecular genetics of HD is also crucial. The gene associated with HD, IT15, was identified in 1993.[3] Individuals with HD have an expanded CAG trinucleotide repeat in one copy of the IT15 gene, usually greater than 39 CAG repeats.[4] Normal individuals have less than 27 CAG repeats in each copy of their IT15 gene.[4] It is important for individuals to understand that an intermediate range of CAG repeats may or may not result in HD for the individual, although it is possible for future generations to inherit an expanded CAG repeat associated with HD.[4] This is due to meiotic instability which results in an expansion of CAG repeats from one generation to the next.

In HD, a CAG trinucleotide repeat of greater than 26 has been associated with meiotic instability.[4] An expansion is more likely to occur when paternally inherited.[5] The CAG repeat size is loosely associated with age at onset where the larger size repeat is associated with a younger age at onset.[6] This association can not be used to predict age at onset for specific individuals, since individuals can have the same CAG repeat size but have different ages of onset.[6]

Abbreviations Used:

HD	Huntington's Disease
PCR	polymerase chain reaction

THE DNA TEST

The next topic for counseling is the molecular testing. The direct gene test uses polymerase chain reaction (PCR) to determine the CAG repeat length in each copy of the IT15 gene.[4] This test is highly accurate. Direct gene testing can determine whether an individual has a CAG repeat length in the HD range, in the indeterminate range, or in the normal range. If a

CAG repeat length in the HD range is identified, that individual will develop HD during his/her lifetime. As mentioned, genetic testing cannot predict age at onset, severity of symptoms or disease progression. The children of an individual with a CAG repeat length in the HD range are at 50% risk for the condition. If a CAG repeat length in the indeterminate range is identified, then the exact risk to develop HD may be unknown. The children or grandchildren of an individual with a CAG repeat length in the indeterminate range may be at risk for HD due to an expansion of the repeat. If the CAG repeat length in both copies of the IT15 gene is normal, the individual and his/her children are not at risk for HD. A review of possible results is crucial to ensure that each individual understands the implications of each result as well as the possibility of an indeterminate result.

MOTIVATIONS FOR TESTING

The counselor should explore the individual's motivations for testing. Individuals who seek presymptomatic testing for HD fall into one of four general categories: young adults, older at risk parents, newly at risk individuals and clinically affected individuals.[8] Young adults often use testing information to make life decisions. Older at risk parents may not want to know their status, but consider testing to clarify the risk of their adult children. Newly at risk individuals have not been previously aware of their at risk status. Therefore they often pursue testing to reassess their plans for the future. Some individuals have not yet realized they are clinically affected and present for presymptomatic testing. These individuals may have an idea that they are affected and may be looking for confirmation while others are surprised. Identifying an individual's motivations for testing allows the genetic counselor to determine whether those motivations are based on accurate information and expectations. In addition, the genetic counselor is able to assess and discuss the potential impact of testing.

> *Presymptomatic testing for HD has a profound effect on the individuals tested. It is crucial that they receive genetic counseling and support services from professionals knowledgeable about both the genetic test itself and the ramifications of testing.*

IMPACT OF DETERMINING HD STATUS

Prior to testing, genetic counselors discuss the effect of test results on life decision making. Since many individuals pursue testing in order to use the results for future planning, it fol-

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lows that testing has a potentially enormous impact. Individuals may choose not to have a family because they are concerned about the risk for HD in children and/or they are concerned about how HD will affect their parenting abilities. Additionally, they may alter their financial and educational decisions. This discussion should help the individual realize how positive or negative test results will impact his/her decisions.

Since presymptomatic testing can have significant psychological ramifications,[9,10] the counselor should discuss possible ramifications prior to testing. Often an individual has already decided whether they will develop HD. A test which reveals an unexpected result may cause psychological distress. In addition, some individuals who test positive become extremely anxious and hypervigilant for symptoms.[8] Individuals have attempted suicide after learning their HD status. This discussion allows the individual to assess the possible psychological ramifications of testing. In addition, it allows the genetic counselor to explore with an individual the possible effects of determining their status as well as the risk for adverse psychological events including suicide.

Since determining the HD status of an individual can affect relationships with family members and friends,[11,12] a discussion of how relationships may be affected is warranted. Individuals who test positive may be concerned about their spouse's response to the result. Parents may feel guilty when their adult child tests positive, even though they had no control over the outcome. When one sibling tests negative, s/he may feel guilty for escaping HD, while the sibling who tested positive did not. Relationships with friends can change as well. Friends may feel as though they do not know what to say or how to be supportive. Some friendships may be strengthened while others are lost. At the end of this discussion, an individual should understand the impact testing has on relationships. In addition, a plan to deal with possible effects can be constructed.

Genetic counseling must include a discussion about the possibility of discrimination by insurance companies and employers. Health care providers and patients have been concerned about the potential for denial, cancellation or refusal to renew insurance policies based on genetic test results. In addition, concerns have been raised about varying premiums or conditions based on genetic information. The true risk for this discrimination is unknown, but most individuals who opt for testing pay for services out-of-pocket to reduce this risk. In addition, concerns have been raised about the risk for job loss related to genetic information. Again, the true risk is unknown at this time, but individuals should understand the potential risk prior to determining their HD status.

Despite preparation and careful decision making, the initial impact of a test result can be overwhelming. Some individuals may feel as though they are in a state of shock. It is the role of the genetic counselor to discuss possible reactions to test results as well as ways to deal with this initial reaction. At the time of disclosure, genetic counselors must determine whether an immediate mental health counseling referral is appropriate. Many individuals do not require referral although follow-up with a genetic counselor and/or a mental health counselor is recommended.

CONCLUSIONS

Presymptomatic testing for HD has a profound effect on the individuals tested. It is crucial that they receive genetic counseling and support services from professionals knowledgeable about both the genetic test itself and the ramifications of testing. Presymptomatic testing is known to impact specific areas of an individual's life. Therefore a detailed discussion of these issues should be undertaken prior to testing. As our ability to perform new genetic tests has grown, we have seen that testing can bring both substantial benefits and concerns. The examination of these possible effects allows individuals to determine whether they will truly benefit from testing.

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Rhode Island Primary Care Physicians' Attitudes Toward Genetic Testing for Breast Cancer

Frances M. Alexakos, CAGS

What do Rhode Island primary care physicians think of the genetic tests for breast cancer? A survey was distributed to a stratified random sample of 300 licensed physicians who self-reported in one of the following specialties: general practice, internal medicine, pediatrics, obstetrics/gynecology, and family practice; 162 physicians (54%) responded.

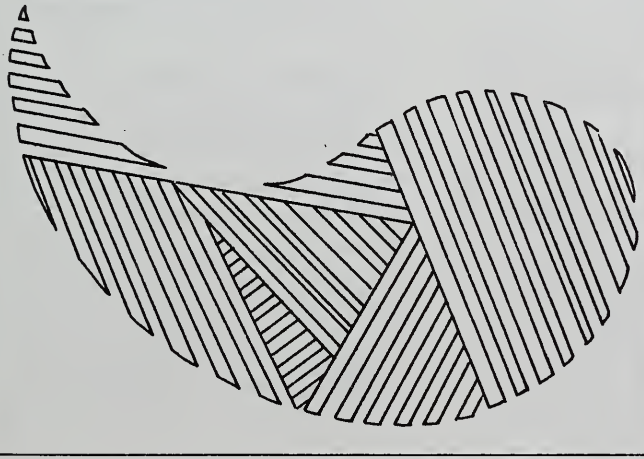
The questionnaire presented statements: respondents could strongly agree, agree, disagree, strongly disagree (Likert Scale). Grouping the responses into two categories - agree and disagree - here are physician responses to key statements.

A majority of physicians agreed with the following statements:

1. Doctors should order a genetic test for a patient whose mother or sister had pre-menopausal breast cancer. Agree: 67%
2. Family members of a patient who has been diagnosed with a defective BRCA1 gene for breast cancer should be informed of their genetic predisposition to breast cancer. Agree: 81%
3. A primary care physician's role should include the provision of genetic counseling. Agree: 76%
4. A person who knows her genetic predisposition to breast cancer is subject to added emotional stress. Agree: 90%

A majority of physicians disagreed with the following statements:

1. Genetic tests accurately predict the possibility of breast cancer. Disagree: 60.8%
2. Health Maintenance Organizations should have a right to require genetic testing for their subscribers as a part of disease prevention. Disagree: 89%
3. Insurance companies, which pay for genetic testing, should have access to the results. Disagree: 85.7%



These questions were abstracted from a PhD dissertation submitted at Salve Regina University.

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Screening and Management of Hereditary Breast Cancer

Robert D. Legare, MD

Breast cancer remains the most common cancer diagnosis in American women and the second leading cause of cancer death. Risk factors for the development of breast cancer include age, family history, exogenous and endogenous hormonal factors and environmental factors such as alcohol consumption. The lifetime risk is estimated to be one in nine, or 12.5%. Age remains the most important risk factor. For example, the risk of developing breast cancer at the age of 50 (1 in 400) is tenfold greater than that at age 30 (1 in 4200).[1] Through a better understanding of genetic and environmental factors which predispose to the development of breast carcinoma it may be possible to employ more effective screening programs as well as therapeutic and preventive interventions. Moreover, determining how a specific risk factor predisposes to breast cancer may be pivotal in understanding the basic process of tumorigenesis.

The majority of breast cancer is sporadic, occurring in women without a family history of breast cancer. Approximately 15-20% of breast cancer is associated with some family history. In general, a twofold to threefold increase in the risk of breast cancer development has been associated with breast cancer in a mother or sister.[2] Importantly, a woman's risk of breast cancer is strongly related to the number and type of relatives affected as well as the age at which these relatives were diagnosed.[2] Presumably, this familial clustering is a result of multiple, relatively weaker genetic influences, single cancer susceptibility genes with low penetrance and shared environmental risk factors. Only 5-10% of breast cancer is thought to be due to the inheritance of a single, highly penetrant autosomal dominant mutation in a single cancer susceptibility gene such as BRCA1 or BRCA2.

Abbreviations Used:

BCT	breast conservation therapy
CASH	Cancer and Steroid Hormone Study
NSABP P1	National Surgical Adjuvant Breast and Bowel Project chemoprevention trial

Elizabeth Claus at Yale, utilizing the Cancer and Steroid Hormone Study (CASH) data set, predicted the presence of a rare autosomal dominant allele leading to increased susceptibility to breast cancer, including early onset breast cancer.[2] Subsequently, BRCA1[3] and BRCA2[4] have been isolated through linkage analysis and extensively characterized. Germline mutations in either gene increases the likelihood that cancer will develop during the lifetime of the individual. BRCA1 accounts for approximately 20-40% of familial breast cancer syndromes. BRCA1 is a tumor suppressor gene whose phenotype is inherited in an autosomal dominant fashion through the maternal or paternal line. The gene is located on the long arm of chromosome 17 and codes for a large protein of 1,863 amino acids.[3] BRCA1 co-localizes and physically associates with RAD51 and may be important in DNA repair and cell cycle kinetics.[5] Mutations in BRCA1 can occur almost anywhere in the coding sequence as well as promoter region, leading to truncation of the encoded protein and inactivation of critical functions. Over 500 different mutations have been reported throughout the 22 coding exons, including frameshift, missense and nonsense mutations. The majority of mutations are unique and limited to a given family. Due to the founder effect, some mutations have a given frequency in certain populations. For example the 185delAG and 5382insC mutations are found in 1.05% and 0.11% of the Ashkenazi Jewish population, respectively[6] and the C4446T mutation has been characterized in French Canadian families.[7] The lifetime risk of breast cancer associated with germline BRCA1 mutations varies from 50-85%. Moreover, second primary breast cancers occur in 40-60% of women, higher than the 0.7-1.0% risk per year of second primary breast carcinoma in the general population. The lifetime risk of ovarian cancer in women who harbor such a mutation is between 16-44%.[6] There is also a possible increase in the risk of prostate and colon cancer.

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BRCA2 is a second tumor suppressor gene located on the long arm of chromosome 13 and codes for a large, highly charged protein of 3,418 amino acids.[4] Mutations in this gene are responsible for approximately 10-30% of familial breast cancer. The mutant allele is similarly inherited through the maternal or paternal line, is expressed in an autosomal dominant fashion and leads to truncation of the encoded protein. BRCA2 also appears to co-localize with RAD51 and may be important in DNA repair and cell-cycle kinetics.[5] Several founder mutations have been identified within BRCA2 including the 6174delT mutation present in 1.36% of the Ashkenazim[6], 8765delAG in the French Canadian population[7] and the 999del5 mutation in the Icelandic population.[8] Studies of the Icelandic population have shown that this mutation is found in 7.7% of unselected female breast cancer patients and in an estimated 0.6% of the general population.[9] The lifetime risk of breast cancer in women who harbor a germline BRCA2 mutation equals that of those harboring a BRCA1 mutation, although early onset breast cancer is less common.[9] Lifetime risk of ovarian cancer in women harboring a germline BRCA2 mutation ranges from approximately 10-20% and there is an increased risk of male breast cancer.[10] There is also an increased risk of prostate, laryngeal and pancreatic cancers in those harboring BRCA2 mutations, but the magnitude of this effect is unknown.[10,11]

While BRCA1 and BRCA2 contribute 30-70% to the cause of hereditary susceptibility to breast cancer, other genes have been identified including germline TP53 mutations in the Li-Fraumeni Syndrome.[11] This syndrome is characterized by bone and soft tissue sarcomas, leukemia, brain tumors, adrenocortical carcinomas as well as breast carcinoma, which is the most common malignancy in adults. Li-Fraumeni syndrome is associated with a 50% risk of carcinoma by age 35, and a 70-90% lifetime risk of cancer development for men and women, respectively.[11] Germline mutations in the PTEN gene are associated with Cowden's Disease, characterized by multiple hamartomas including papillomas and trichilemmomas, thyroid disease, uterine fibroids, macrocephaly, mental retardation and breast cancer.[12] Both benign and malignant breast tumors occur in Muir-Torre syndrome, a syndrome related to hereditary nonpolyposis colon cancer.[13] Peutz-Jegher syndrome , associated with germline mutations in the STK11 gene and characterized by benign gastrointenstinal polyps and abnormal pigmentation, is also associated with an increased risk for breast cancer including early onset bilateral disease.[14] It is likely that mutations in these genes account for less than 2% of hereditary breast carcinoma. Therefore, between 30-70% of the hereditary susceptibility to breast cancer is due to yet uncharacterized genes. A third locus on chromosome 8p12-p22 has been shown by linkage analysis and loss of heterozygosity to contain a putative third breast cancer susceptibility gene around marker D8S505.[15]

Women thought to be at high risk for a familial breast cancer syndrome who elect to further investigate these issues should be referred for formal cancer risk assessment. Ideally, this would include a multidisciplinary team of specialists composed of genetic counselors as well as physicians and surgeons specifically trained and focused upon clinical cancer genetics.

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Features of hereditary cancer syndromes which would increase one's index of suspicion for a patient harboring a germline mutation would include: clustering of cancer in families, tumors that are multifocal or bilateral, multiple primary neoplasms in the same individual, male breast cancer and cancers developing at unusually early ages. The presence of one or more cases of ovarian cancer in a family with breast cancer substantially increases the probability of detecting a germline mutation. Genotype-phenotype relationships are actively being investigated but no clear data is currently available for BRCA1 and BRCA2 mutations. The histopathology of breast carcinoma of BRCA1 breast cancer patients has generally been shown to be unfavorable. Specifically, these tumors are characterized by high grade, aneuploidy and an increased S-phase fraction. Such primary tumor characteristics have been associated with more aggressive clinical behavior and worse prognosis in the general population of women with breast cancer.[16] There has been conflicting data on the prognosis of breast cancer in those harboring a germline mutation versus the general population. It is likely that survival for carriers of BRCA1 and BRCA2 mutations is similar to or worse than that of breast cancer patients in general.[16,17] Based on histopathology, it is surprising that survival of BRCA1 patients is not significantly more unfavorable.[17] One possible contributing factor to this discrepancy may lie in the apparent increase in medullary carcinoma, a prognostically favorable subtype, in diagnosed BRCA1-associated breast cancer cases.[17]

Women thought to be at high risk for a familial breast cancer syndrome who elect to further investigate these issues should be referred for formal cancer risk assessment. Ideally, this would include a multidisciplinary team of specialists composed of genetic counselors as well as physicians and surgeons specifically trained and focused upon clinical cancer genetics. This team should also include psychiatrists, psychologists and social workers.

Several models may be utilized to estimate an individual's risk of developing breast cancer. The Gail model[18] calculates breast cancer risk using risk factors such as age, hormonal factors, number of first degree relatives with breast cancer and number of previous breast biopsies. This model does not incorporate a family history of ovarian cancer, age at onset of breast cancer or breast cancer diagnosis in paternal relatives and, therefore, could significantly underestimate the risk for BRCA1 and BRCA2 mutations in carriers and overestimate the risk for their nonaffected relatives. The Gail model was utilized to assign risk and, therefore, determine eligibility for the National Surgical Adjuvant Breast and Bowel Project chemoprevention trial (NSABP P1), which reported a 50% reduction in the risk of breast cancer in a population of high risk women with a median follow-up of 48 months.[19] This cohort is currently being assessed for BRCA1 and BRCA2 carrier status and the effect of tamoxifen on this cohort will subsequently be reported. The Claus model[2] estimates breast cancer risk based on family history alone and includes age at onset and paternal relatives with breast cancer but excludes other malignancies such as ovarian cancer. Like the Gail model, the Claus model is best used for estimating risk in moderate risk families and can significantly underestimate risk for germline mutation carriers and over-estimate risk for their nonaffected relatives.

Several statistically based models have been specifically developed to estimate the risk of BRCA1 and BRCA2 carrier status based on personal and family medical history.[20,21] These models have been developed to aid in determining whether an individual's probability of harboring a germline mutation is sufficient to merit consideration of genetic testing. According to the American Society of Clinical Oncology, genetic testing should not be performed unless the pretest probability of carrier status is at least 10%.[22] Women who decide to undergo genetic testing require pre-test and post-test counseling to ensure the highest level of informed consent and long term psychological well-being. It is critical to remember that, if genetic testing is desired and appropriate, it should preferentially occur in an affected individual. Testing an unaffected individual is only interpretable in the setting of a positive result for the presence of a germline mutation.

It is critical to remember that, if genetic testing is desired and appropriate, it should preferentially occur in an affected individual.

There are few evidence-based recommendations regarding the optimal cancer surveillance in individuals who harbor BRCA1 or BRCA2 mutations. A task force with expertise in medical genetics, oncology, primary care, gastroenterology, and epidemiology convened by the Cancer Genetics Studies Consortium and organized by the National Human Genome Research Institute published recommendations for the follow-up care of individuals known to carry BRCA1 and BRCA2 mutations or those who have a substantial likelihood of being mutation carriers.[23] Monthly breast self-examination to establish a regular habit and to allow familiarity with the normal characteristics of breast tissue as well as instruction on self-examination is recommended starting at an early age (e.g., by age 18-21 years). An annual or semi-annual clinician examination, preferably performed by a breast surgeon or clinician proficient in breast examination, is recommended beginning at age 25 to 35 years. Annual mammography is suggested, beginning at age 25 to 35 years. Ultimately, the program for the surveillance employed should be determined by the patient after proper review and counseling regarding the present limitations of data and possible implications/complications from any screening intervention. For example, mammography in younger women is less sensitive than older women and could lead to unnecessary diagnostic procedures. No data is currently available on radiation risk of early mammography on BRCA1 or BRCA2 mutation carriers.[23]

As mentioned, data regarding the risk reduction associated with tamoxifen in carriers of BRCA1 and BRCA2 mutations is currently lacking but will be analyzed from the NSABP P1 data set after testing of this cohort is complete. Germline carriers who would have met the entry criteria for this trial would certainly be reasonable candidates for tamoxifen as a means for risk reduction. Based on the increased risk of breast cancer for mutation carriers, tamoxifen may be elected by certain individuals for risk reduction outside of the NSABP P1 criteria.

Prophylactic bilateral mastectomy is another option to decrease the risk of breast cancer in women with a strong family history of the disease. Simple (total) mastectomy is more effective than subcutaneous mastectomy, which preserves the areola and a small amount of ductal tissue beneath them. There are no prospective data on the use of prophylactic mastectomy in any cohort of high risk women, including mutation carriers. A decision analysis predicts a modest (2.9 to 5.5 year) gain in life expectancy with the procedure.[24] A recent retrospective series of 639 women with a family history of breast cancer who underwent bilateral prophylactic mastectomy noted a risk reduction in the incidence of breast cancer of at least 90%.[25] While this trial is retrospective, the study design was quite strong, and a prospective, randomized trial with a surgical and nonsurgical arm will likely not be forthcoming as it would be unacceptable to most women. For women who elect prophylactic bilateral mastectomy, continued surveillance with breast examination is recommended after surgery.

Modifiable nongenetic risk factors may also be addressed as a means of potential risk reduction; e.g., decreasing alcohol consumption[26] and possibly fat consumption. Lifestyle modifications which are generally thought to be beneficial, but which are not clearly associated with a risk reduction in breast cancer, include high fiber diet and exercise.

Individuals who are mutation carriers and develop breast cancer may chose from the established options for local and systemic therapy similar to that of their non-carrier counterparts. A recent retrospective study evaluating overall survival in 30 BRCA1 and 20

BRCA2 breast cancer patients with a median follow-up of 9.8 years and 7.5 years, respectively, noted a normal skin reaction to radiotherapy and a similar prognosis when compared with sporadic breast cancer cases. However, only 6 mutation carriers received breast conservation therapy (BCT). While other studies suggest BCT including radiotherapy in mutation carriers is safe, there is no prospective data and extremely limited retrospective data.[16] The effect of radiotherapy scatter on the contralateral breast is also of unclear significance in this population of women at high risk for second primary breast tumor.

Cancer genetics and its applications to the clinical venue are currently in their infancy. It is hoped that, through collaboration, prospective data on counseling, screening, surveillance and treatment will be forthcoming to better serve this high risk population.

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National Initiatives: Management of Heart Disease and Stroke

Edward Westrick, MD, MS

Cardiovascular disease and stroke are respectively the first and third leading causes of death in this country. They are caused primarily by a common pathophysiologic process and therefore share common risk factors, some of which are modifiable. Risk factor modification refers to our efforts to prevent heart attacks and strokes. In this column I will lay out a causal model of cardiovascular disease and discuss secondary prevention strategies. Since Rhode Island Quality Partners (RIQP), and all Peer Review Organizations (PROs), will be investing a majority of their efforts over the next few years in these conditions, I will outline for you the measurement issues and opportunities for improvement that we will likely be working on together.

Abbreviations Used:

AAHP	American Association of Health Plans
AHCPR	Agency for Health Care Policy and Research
AHQA	American Health Quality Association
AMA	American Medical Association
APhA	American Pharmaceutical Association
HCFA	Health Care Financing Administration
HCQIP	Health Care Quality Improvement Program
JCAHO	Joint Commission on the Accreditation of Healthcare Organizations
NCQA	National Committee for Quality Assurance
PRO	Peer Review Organization
RIQP	Rhode Island Quality Partners

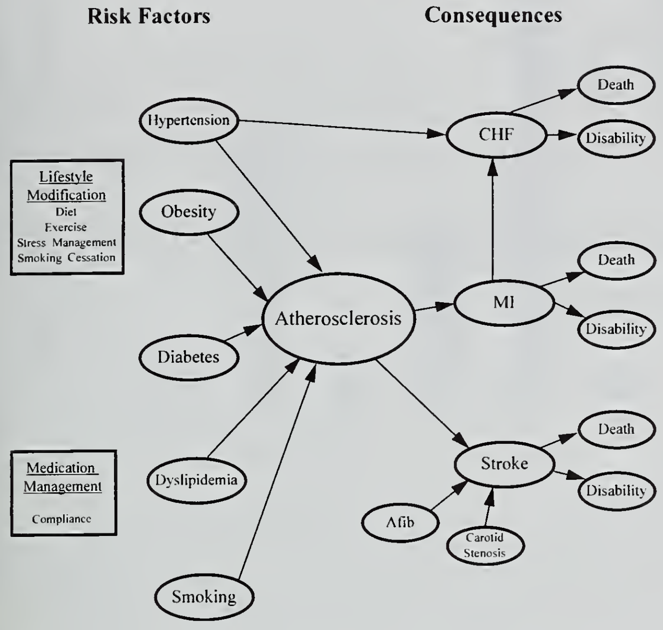
The figure takes the form of what some folks would call a "causal model." This model uses arrows to designate statistical associations, some of which may be causal. There may be a more appropriate description of this kind of model that I remember from residency training. During morning report and on teaching rounds, Phil O'Dowd would ask us to explain our "cartoon models" representing our understanding of what was going on clinically. So think of the figure as a cartoon model or causal model, whichever you prefer.

Both myocardial infarction and stroke are mediated by the pathophysiologic process atherosclerosis. Myocardial infarction can lead to congestive heart failure, disability or death. Both congestive heart failure and stroke can lead to disability or death. Atherosclerosis has many known risk factors some of which are modifiable. The non-modifiable ones include family history of early cardiovascular disease, age, and gender. The modifiable ones include dyslipidemia, hypertension, smoking, and obesity. Diabetes mellitus is a special sort of risk factor. Glycemic control can be achieved but it is not clear that better control reduces risk for cardiovascular disease. We must therefore classify diabetes mellitus as a non-modifiable risk factor with respect to cardiovascular disease at this time. However, glycemic control does reduce risk for microvascular disease (nephropathy, retinopathy, and neuropathy) so we must classify diabetes mellitus as a modifiable risk factor with respect to these diseases. Notice that stroke has additional risk factors not common to myocardial infarction including atrial fibrillation and carotid stenosis. And finally, hypertension is a risk factor for congestive heart failure directly, in addition to having an influence presumably mediated via atherosclerosis. There are many complexities in the associations that cannot be represented in this cartoon.

The far left side of the figure displays the components of clinical management that are part of modifying the risk factors. It turns out that the lifestyle modifications and medication management issues are largely relevant to all of the atherosclerotic disease risk factors. Lowering body weight through diet and exercise lowers blood pressure, improves the serum lipid profile, and can enhance insulin receptor sensitivity. There are lifestyle modification goals and medication compliance issues that are common to these risk factors. These conditions are relatively asymptomatic and their associated treatments can seem like a burden to patients. It is difficult for patients and sometimes providers to appreciate the value of prevention.

The national health care quality improvement movement has recognized that these are important diseases because of their consequences, costs, and sheer volume in the population. Efforts are being made by accreditors, regulators, and payers to improve practices in these diseases. Part of improving practice

Cardiovascular
Risk Factors and Consequences



patterns requires the measurement of these practices. Here is how some of these stakeholders will be measuring quality over the next few years.

HCFA's Health Care Quality Improvement Program (HCQIP) is implementing a national initiative in stroke, acute myocardial infarction, congestive heart failure, and diabetes mellitus in the next three year contract cycle for the PROs.[1] Measurement of performance will be accomplished using common indicators, abstraction processes, and analyses. This commitment should lead to increased comparability. For congestive heart failure we anticipate that ACE inhibitor and diuretic prescribing will be measured as well as the assessment of left ventricular ejection fraction. For acute myocardial infarction we anticipate that thrombolytic therapy and early aspirin administration will be measured. After acute myocardial infarction, secondary prevention practices will be evaluated including the use of beta-blockers, aspirin, ACE inhibitors, cholesterol lowering therapy, and smoking cessation counseling in appropriate candidates. In stroke, we anticipate that anticoagulation in atrial fibrillation will continue to be a focus of measurement as well as the use of rapid acting antihypertensives, aspirin and other antiplatelet agents. In diabetes mellitus we anticipate that LDL testing and achievement of LDL goals, glycosylated hemoglobin testing, nephropathy testing, and referral for periodic retinal exams will be measured. In the near future, achievement of blood pressure below 140/90, documentation of foot exams, and glycemia out of control will be added to the diabetes mellitus set of measures.[2]

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The American Heart Association has stated as its goal to reduce coronary artery disease, stroke, and risk by 25% by the year 2008. That is indeed a bold goal. Locally, the RI division has decided to focus on secondary prevention.[3] You can look forward to some excellent educational programs, and special tools in development to help physicians and other clinicians follow the guidelines developed by the American Heart Association a few years ago.

There is a national initiative to improve medication use. The Coalition to Improve Quality in Medication Use was formed last year and is working now to develop a handbook of indicators to measure quality in this area. This coalition, made up of more than 50 national organizations including JCAHO, HCFA, AMA, AHQA, NCQA, AAHP, AHCPR, and APhA, has chosen to focus initially on these same diseases. Notice how medication use accounts for a large proportion of the indicators listed above.

Looking ahead to our next three years working with HCFA we have identified other mandatory topics that relate strongly to medication use issues. In community acquired pneumonia we will be measuring the delay to first dose of antibiotics for hospitalized patients as well as antibiotic coverage for atypical organisms. In the prevention of pneumonia and other respiratory conditions, we will be looking at pneumococcal and influenza immunization.

So you can see that medication use is going to be an important focus of our quality improvement activities. And the vast majority of that work will be done in preventing heart disease and stroke. We look forward to working with physicians, hospitals, health plans, home care agencies, and long term care facilities, as well as state and federal government, associations and societies, our institutions of higher education, accreditors and regulators. It will be nice to look back in 2008 to see how far we've come, and then set some new bold goals.

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Health by Numbers

Rhode Island Department of Health
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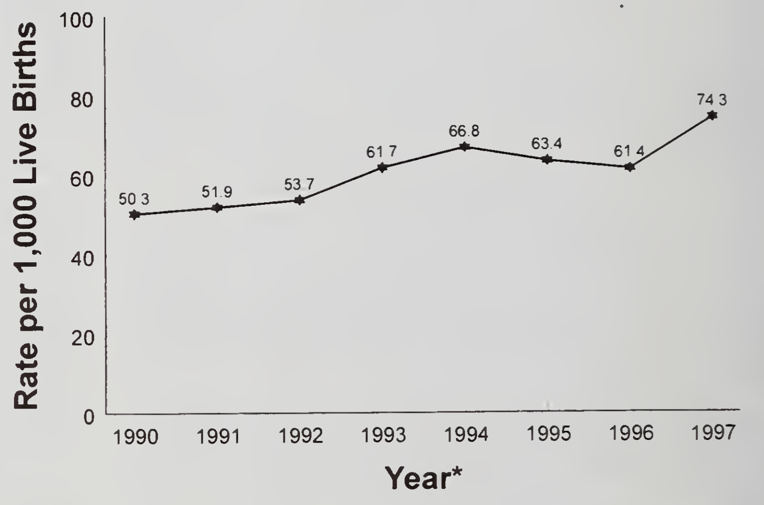
Edited by Jay S. Buechner, PhD, and William J. Waters, Jr., PhD

Congenital Anomalies Among Hospital Newborns

Jay S. Buechner, PhD

Every year in Rhode Island, a number of newborns are born with congenital anomalies. These occur in many organ systems and range from trivial to severe in their impact on the child's health. In this article, congenital anomalies identified during the infant's hospitalization at the time of birth are analyzed, as reported on the statewide hospital discharge database for the period from October 1989 through September 1997.



* Twelve months ending September 30.

Figure 1. Congenital Anomalies per 1,000 Newborns in Rhode Island Hospitals, by Hospital Fiscal Year, October 1989 - September 1997.

Methods

Under licensure regulations, all acute-care hospitals in the state report to the Department of Health a defined set of data items on each inpatient discharged from those hospitals. The data items include information on diagnoses coded in the International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM).[1] Newborn discharges were identified as discharges having a principal diagnosis in the code range V30 - V39. The presence of one or more congenital anomalies was determined by examining the six additional diagnosis fields for codes in the range of 740 - 759. Congenital anomalies of specific organ systems were defined as follows:

Central nervous system	ICD-9-CM 740-744
Circulatory and respiratory systems	ICD-9-CM 745-748
Gastrointestinal system	ICD-9-CM 750-751
Urogenital system	ICD-9-CM 752-753
Musculoskeletal and integumentary systems	ICD-9-CM 749, 754-757
Chromosomal	ICD-9-CM 758
Other	ICD-9-CM 759

Incidence rates for congenital anomalies were computed per 1,000 liveborn infants. Because an infant may have more than one congenital anomaly, the incidence rates for anomalies in specific organ systems may total to more than the overall rate for congenital anomalies. The analysis describes newborns with congenital anomalies born during the eight-year period from October 1, 1989, through September 30, 1997 (hospital fiscal years 1990 through 1997).

Table 1. Most Commonly Reported Specific Congenital Anomalies among Newborns in Rhode Island Hospitals, October 1989 - September 1997.

Congenital Anomaly (ICD-9-CM Code)	Number
Congenital pigmentary anomalies of skin (757.33)	755
Patent ductus arteriosus (747.0)	629
Ventricular septal defect (745.4)	474
Hypospadias, epispadias, and other peniledefects (752.6)	470
Ostium secundum type atrial septal defect (745.5)	402
Undescended and retractile testicle (752.5)	386
Other specified anomalies of skin (757.39)	287
Congenital subluxation of hip, unilateral (754.32)	274
Vascular hamartomas (757.32)	245
Obstructive defects of renal pelvis and ureter (753.2)	182

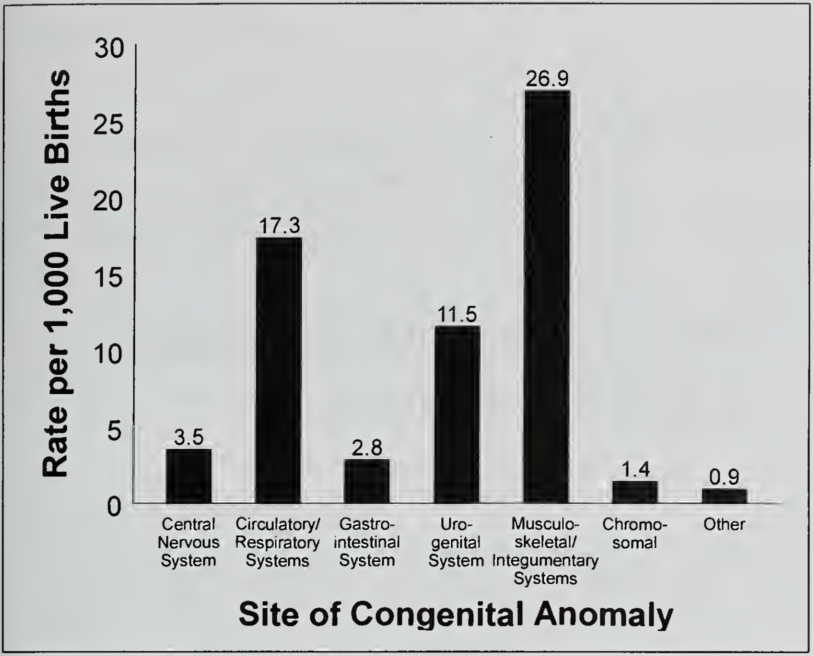


Figure 2. Congenital Anomalies per 1,000 Newborns in Rhode Island Hospitals, by Site of Congenital Anomaly, October 1989 - September 1997.

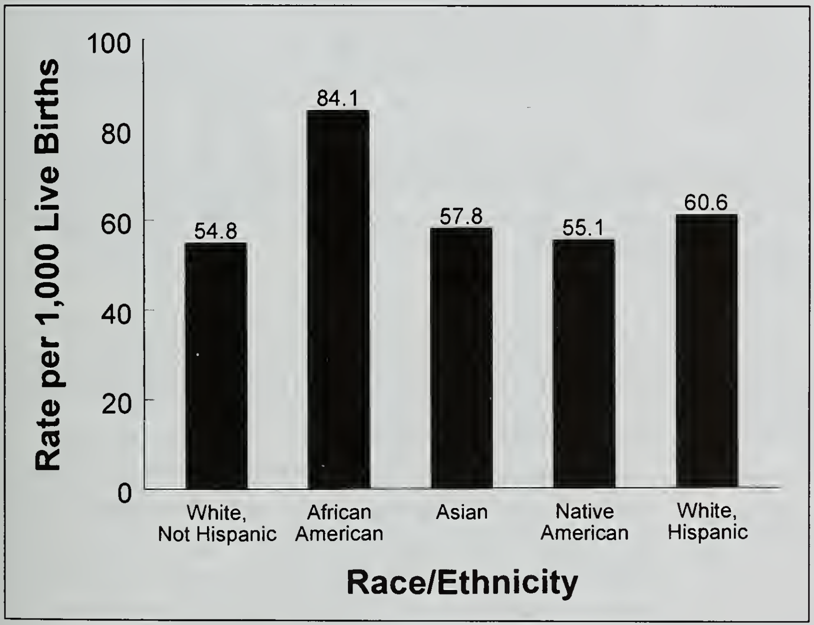


Figure 3. Congenital Anomalies per 1,000 Newborns in Rhode Island Hospitals, by Race/Ethnicity of Newborn, October 1989 - September 1997.

Results

Over the eight-year period studied, approximately 6% of hospital newborns (59.9 per 1,000) were reported as having one or more congenital anomalies. Over time, the incidence rate rose unevenly from 50.3 per 1,000 in fiscal year 1990 to 74.3 per 1,000 in fiscal year 1997, for an overall increase of 47.7%. (Figure 1)

Congenital anomalies were reported in all categories of organ systems, but with greatly varying rates of incidence. Most common were congenital anomalies of the musculoskeletal and integumentary systems, followed by anomalies of the circulatory and respiratory systems and anomalies of the urogenital system. (Figure 2) The most frequently occurring specific congenital anomalies are presented in Table 1.

The rate of congenital anomalies varied with race and ethnicity. All minority groups were reported as having higher rates than for white, non-Hispanic infants. (Figure 3) Rates were greatly elevated for African-American infants, whose rate over the eight-year period was 40.4% above the state average for all race/ethnicity groups and 53.5% above the rate for white, non-Hispanic infants. Approximately 60% of the elevation relative to the white, non-Hispanic population is due to a higher rate of congenital anomalies of the integument. For other minority groups, the elevations were small.

The reported incidence rates for congenital anomalies varied over time and by hospital of birth. For example, the eight-year incidence rate for congenital anomalies of all kinds varied among hospitals from a low of 2.66 per 1,000 to a high of 18.35 per 1,000. Hospitals have also displayed different trends over time in reported incidence rates. Comparing the four fiscal years 1990-1993 with the four fiscal years 1994-1997, all hospitals showed increases, but the increases ranged from minimal (0.4%) to substantial (57.5%).

Discussion

Hospital discharge data are useful for surveillance of the number and types of congenital anomalies among newborns in Rhode Island. Other data sources, such as birth certificate records and laboratory reports, contain information only on specific anomalies. The reliability of the information obtained from hospital discharge data may be called into question, however, because facilities report widely differing incidence rates among newborns. This variability needs to be explored with the hospitals to assure consistent reporting of these events.

Nevertheless, the available data show high and apparently increasing incidence of congenital anomalies among newborns in Rhode Island. The most serious anomalies often require that the child be evaluated, treated, and followed at clinics such as the Child Development Clinic and the Cardiac Clinic at Rhode Island Hospital.

Accurate information on the incidence and prevalence of specific congenital anomalies can help determine the needs for such specialty treatment and help plan for the capacity to meet those needs. Physicians can help by complete and careful reporting of congenital anomalies on birth certificates, in hospital records, and in other reporting systems.

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PUBLIC HEALTH BRIEFING

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

0.08: A Safer Legal Blood Alcohol Limit for Drivers

Nancy Libby Fisher, MMHS

We can reduce alcohol-related traffic crashes.

Experience has shown that lowering the legal blood alcohol concentration (BAC) limit to 0.08 reduces alcohol-related traffic crashes and the fatalities and injuries that result from them.

States that have lowered the legal BAC limit for drivers to 0.08 have experienced a 16% decline in the proportion of fatal crashes involving fatally injured drivers whose blood alcohol levels were 0.08 or higher.[1]

Setting the BAC limit for drivers to 0.08 serves to deter impaired driving. Crash statistics show that even heavy drinkers, who account for a large percentage of drunk driving arrests, are less likely to drink and drive because of the general deterrent effect of a 0.08 BAC limit.

At the same time, lowering the BAC limit to 0.08 will make it possible to convict seriously impaired drivers whose blood alcohol concentrations are now considered marginal because they are at or just over 0.10 BAC.

Virtually all drivers are substantially impaired at 0.08 BAC.

Although outward appearances vary, research shows that the majority of drivers, even experienced drivers, are substantially impaired at 0.08 BAC regarding critical driving tasks. There are significant impairments in braking, steering, lane changing, judgment and divided attention at 0.08 BAC. Studies show that performance decrements in some of these tasks are as high as 60 to 70% at 0.08 BAC.

Abbreviations Used:

BAC blood alcohol concentration

The impairments in reaction time, attention, tracking, comprehension and other skills essential to safe driving increase the probability of a crash. This probability rises significantly after 0.05 BAC and the increase is substantial by 0.08 BAC. There is a gradual increase in risk for being in a crash at each BAC level, but after a driver reaches or exceeds 0.08 BAC the rise is very rapid.

Even when trying to drive carefully, an impaired driver cannot compensate for these reduced abilities, and alcohol affects the ability of individuals to judge whether or not they are impaired.

Lowering the legal BAC limit for drivers to 0.08 does not hurt the hospitality industry.

While there is no "safe" amount of alcohol for drivers, most people can drink moderately and drive legally when the legal limit is set at 0.08 BAC. A 0.08 BAC is not typically reached with a couple of beers after work or a glass or two of wine with dinner (Table 1). Accordingly, per capita alcohol consumption is not measurably affected in states with 0.08 BAC legislation.[2] Surveys show that most people would not drive after consuming 2 or 3 drinks in an hour and believe the limit should be no higher than that. Based on recent polls, 2 out of every 3 Americans favor lowering the limit to 0.08 BAC when they know how much alcohol it takes to reach that level (Table 1).

Table 1. Drinks in body* needed to reach a 0.08 BAC

	Weight in Pounds							
Gender	100	120	140	160	180	200	220	240
Female	2	3	3	4	4	4	5	5
Male	3	3	4	4	5	5	5	6

* Defined as drinks consumed in one hour on an empty stomach.

* A drink is defined as 1.5 ounces of 80 proof liquor or 12 ounces of beer or 5 ounces of table wine.

In support of 0.08

The U.S. Surgeon General's Office, the American Association of Neurological Surgeons, and the American Medical Association have come out in support of lowering the legal BAC for drivers to 0.08 or lower.[2]

In the Meantime ...

With regard to physician interventions to prevent motor vehicle injuries, the U.S. Preventive Services Task Force recommends: "Counseling all patients, and the parents of young patients, to use occupant restraints (lap/shoulder safety belts and child safety seats), to wear helmets when riding motorcycles, and to refrain from driving while under the influence of alcohol or other drugs is recommended (see Clinical Intervention)."[3] [The Rhode Island Department of Health also recommends counseling patients and the parents of young patients to wear helmets when riding bicycles!]

For more information

The Rhode Island Unintentional Injury Prevention Program at the Rhode Island Department of Health is prepared to answer questions you or your patients may have on this subject. Please call Nancy Libby Fisher, Program Manager, or Marilou Freitas, Resource Specialist, (401) 222-4420.

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Nancy Libby Fisher, MMHS, is Program Manager of the Rhode Island Unintentional Injury Prevention Program at the Rhode Island Department of Health.

Judicial Diagnosis

Wrongful Birth and the Measure of Damages in Rhode Island

Gary L. Bahr, JD, LLM

When a child is born with a disability, is the physician liable? Maybe, depending upon the circumstances. A physician who performs a sterilization judged "negligent" may be liable. In February 1997, Rhode Island joined the growing list of states that recognize a tort cause of action for performing a sterilization procedure in a negligent fashion. In *Emerson et al. v. Magendantz, MD, et al., 689 A.2d 409* (R.I. 1997), the Supreme Court of Rhode Island was unanimous, two justices with two justices concurring, on the existence of the "cause of action," but divided, two to two, on the proper "measure of recovery." (Chief Justice Weisberger, joined by Associate Justice Lederberg, wrote the opinion on the existence of the cause of action; Associate Justices Bourcier and Flanders concurred. The Chief Justice also wrote for Justice Lederberg and himself on the damage recovery; Associate Justices Bourcier and Flanders dissented in an opinion written by Justice Bourcier.)

The facts of the case were as follows: Having had one child, the Emersons decided, based on financial reasons, that they did not wish to have other children. Mrs. Emerson

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consulted with the defendant gynecologist about a sterilization procedure. In January 1991 the defendant performed a surgical tubal ligation upon Mrs. Emerson. In May 1991 her obstetrician determined that Mrs. Emerson was pregnant. In January 1992, a daughter was born with alleged congenital defects. Mrs. Emerson then had a second tubal ligation.

Chief Justice Weisberger noted that only Nevada has declined to recognize a cause of action for negligently performing a sterilization procedure and the subsequent conception or birth of a child. Thirty-five other jurisdictions have recognized this tort for negligently failing to properly perform a sterilization on either the husband or the wife. Depending on the jurisdiction, the action might be referred to as wrongful conception or wrongful birth, and would be brought by one or both of the adult parties involved. If the action is brought by the child, it is called an action for wrongful life.

In responding to the second question concerning the measure of damages, the Chief Justice divided the thirty-five jurisdictions into three groups. The first group - thirty states - grant a limited form of recovery. The second group - three states - allow recovery for the cost of rearing the child, letting the court offset against the cost of child-rearing the economic or emotional benefits that the parents gain through having a healthy child. The third group - two states - do not allow this offset.

The states in the first, limited recovery, group often compensate for medical expenses for the costs of the first, ineffective, sterilization; the costs of the pregnancy in medical and hospital bills; and the costs for the second attempt at sterilization and for lost wages. This group would generally allow for medical costs of prenatal, delivery, and postnatal care. Some of the states would allow recovery for the emotional distress and for loss of consortium caused by the unwanted pregnancy.

The Chief Justice and Justice Lederberg adopted a limited form of the limited recovery rule. For the wrongful birth of a healthy child, the parents would be allowed recovery of medical and hospital expenses for the ineffectual sterilization procedure, the pregnancy, later sterilization procedure, prenatal care, delivery, and postnatal care; lost wages; and spousal loss of consortium caused by the unwanted pregnancy. They refused to allow recovery for the emotional distress caused by the birth of a healthy, but unwanted, child.

For the wrongful birth of a child with congenital physical or mental disabilities or both, these two justices adopted a more expansive form of recovery. Following the case of *Fassoulas v. Ramey, 450 So.2d 822 (Fla.1984),* they indicated that in these cases they would allow special medical and educational expenses above the normal costs of rearing the child.

They then added that when the physician has notice from the parents or reason to know from professional statistical information that there is a reasonable expectation of the birth of a child with either a physical of mental handicap, the liability expands again. Under either of these conditions, the total cost of the child's rearing should be recoverable. They added that the extraordinary costs recoverable could extend beyond the child's majority. A possible offset would be governmental or other agency contributions toward the child-rearing costs. They seem to say that the emotional distress of the parents should also be compensable, but it is not clear whether this applies to the congenital disabilities situation with notice or without notice.

The two justices who dissented on the measure of recovery would authorize a more expansive recovery to include all damages that are reasonably foreseeable and proximately caused by the negligent doctor. These justices relied on *Burke v. Rivo, 406 Mass. 764, 551 N.E.2d 1 (1990).* They would allow recovery for pain and suffering of the wife flowing from the pregnancy, birth, and second sterilization and the expenses of caring for any other children in the family during the disability of the mother. The biggest differences occur in the allowance of recovery for emotional distress and the costs of raising the child if the no-more-children decision had been taken for economic reasons. They reject the notion that the joy of having the child offsets the costs of rearing. Only if the no-more-children decision is made for eugenic or therapeutic reasons and a normal, healthy baby is born would they back away from awarding the cost of rearing. The jury could reduce the award by any beneficial value brought to the parents by the child's birth.

The views of Chief Justice Weisberger and Justice Lederberg on the damages recovery level seem to be based on potential factual differences in the cases relating to culpability of the physician. For the two dissenters on damages, medical malpractice is medical malpractice is medical malpractice.

There was no majority opinion or view articulated in the *Emerson* case. Since this decision, Associate Justice Goldberg has assumed her seat upon the Rhode Island Supreme Court. Until the court decides another wrongful birth or similar case, the would-be plaintiffs, the medical and legal communities, and the Superior Courts must live with some uncertainty.

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Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	April 1998	12 Months Ending with April 1998		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	225	3,160	319.1	4,037.5
Malignant Neoplasms	202	2,505	253.0	7,103.5**
Cerebrovascular Diseases	41	667	67.4	659.5
Injuries (Accident/Suicide/Homicide)	39	335	33.8	6,388.0***
COPD	23	459	46.4	312.5

Vital Events	Reporting Period		
	October 1998	12 Months Ending with October 1998	
	Number	Number	Rates
Live Births	1,166	13,376	13.5*
Deaths	787	9,722	9.8*
Infant Deaths	(7)	(103)	7.7#
Neonatal deaths	(6)	(82)	6.1#
Marriages	473	7,558	7.6*
Divorces	308	3,197	3.2*
Induced Terminations	335	4,620	345.4#
Spontaneous Fetal Deaths	75	913	68.3#
Under 20 weeks gestation	(69)	(835)	62.4#
20+ weeks gestation	(6)	(78)	5.8#

* Rates per 1,000 estimated population # Rates per 1,000 live births

**Excludes one death of unknown age

***Excludes three deaths of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 990,225

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

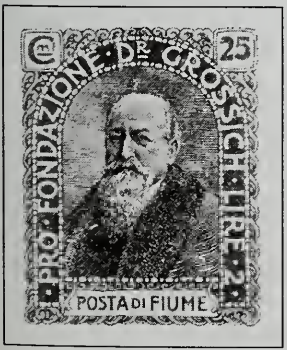
Philately in Medicine

John Tierney

The Introduction of Tincture of Iodine Sterilization

Antonio Grossick (1849-1926) [Fiume, 1919, Scott B 16], who was born in Dragaccio, Italy, studied medicine at Vienna University, graduating in 1875.

Although Fiume City was Hungarian territory in the late 19th century, many Italian immigrants had settled there; and the Italian-born Grossick went to Fiume City to accept the position of Chief of the Surgery Division at the City Hospital.



In 1908, Dr. Grossick invented the application of tincture of iodine as a method of sterilization. He presented the method with many clinical cases; soon it became the "Grossick Method."

A 10%-12% tincture of iodine was applied before surgery and once again after surgery. The tincture has penetrating and sterilizing effects on dried skin. Although perfect sterilization could not be achived by this method, it was effective in the solidification of germs. The merit of the method is that it can be done easily and completed in a short period of time.

Source: Grossick A. "Eine neue sterilisierungs methode der haut bei operationen." *Zentralblatt für Chirurgie* 1908;35:1289-92.

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Ninety Years Ago

[May, 1909]

In "A Case of Early Maternity," R. H. Carver, MD, reported the tale of a 10-year old new mother. Elizabeth Drayton, born in the local almshouse on May 24, 1847 ("so says the record of the old family Bible belonging to that establishment"), gave birth to a child. The 15 year-old father "... belongs to the state of Maine." The writer held high hopes for the newborn Hodges White Drayton ("a beautiful little fellow, with bright blue eyes (and although it is said that a man with a blue eye was never known to command a 74-gun ship), yet to all human appearances he has the essential elements in him to make a great man.") Notwithstanding those hopes, Hodges grew up to serve a term in the New Bedford House of Correction.

Frederick N. Brown, MD, physician for the past 10 years to the Home for Aged Women in Providence, offered "Some Observations Upon Old Age." He listed three main categories of diseases of old age: cardiac and circulatory, digestive, and inanition. Even while conceding that "[old age] is an uphill fight that can eventually have but one ending," the author suggested remedies: " Acid drinks are often useful and unusually gratifying." In particular, he praised the remedial powers of alcohol: "I believe that alcohol in some of its more palatable forms is a sheet anchor in the condition of old age, generally speaking, both for its stimulation and its oxidizable qualities in the tissue.....Especially is alcohol useful for [treating] pneumonia."

Fifty Years Ago

[May, 1949]

In "Acute Diverticulitis of the Cecum," Anthony Migliaccio, MD, reported on 6 cases where the initial preoperative diagnosis of appendicitis was wrong. Only during surgery did physicians recognize the diagnostic error. In one 30 year-old man, Dr. Migliaccio noted, "During the operation, diverticulitis was considered, but no one in the room had ever heard of its occurrence in the cecum, therefore we reasoned that this case represented an early malignancy, and concluded that this patient had been fortunate in having some inflammatory reaction around the carcinoma." After a subsequent right colectomy, the physicians found no malignancy. "At the second operation we marveled at the decrease in the size of the tumor and attributed it to the disappearance of the inflammatory reaction resulting from the previous short circuiting operation. We were chagrined, when on opening the specimen, no tumor could be demonstrated."

Julius C. Early, MC, USN, discussed "Some Aspects of Aviation Medicine" (from a presentation before the midwinter meeting of the Rhode Island Medical Society, at Quonset Naval Air Station). He traced aviation medicine to the first recorded instance where flyers took supplementary oxygen with them. In 1875, the French meteorologist Tissandrier and two companions rose 28,820 feet in a balloon. They had decided to use the oxygen only if they felt the need. Unfortunately, nobody felt the need; and Tissandrier's companions died of anoxia. Paul Bert, a French physiologist who studied the physiology and physics of high altitudes, concluded that at 10,000 feet oxygen would be decreased to 90%, but the pilot would feel no symptoms. At 15,000 feet oxygen saturation would fall to 83%; the pilot would be impaired. Bert recognized that, "It is not enough that a pilot just remain reasonably conscious."

Henry D. Locke, PhD, the Research Director of Liberty Mutual Insurance Company, discussed "What Insurance Companies Are Doing and Can Do To Extend Medical Care in New England" (from a presentation at a meeting sponsored by the Council of New England State Medical Societies). President Truman's Secretary of HEW Oscar Ewing had proposed a national goal of 60% insurance coverage; Locke reported that New England, where 57.5% of the population had hospital insurance, was close. In Rhode Island, 83.7% of the population had hospital insurance - the highest in New England. Maine, with 37.3% coverage, was lowest. Coverage for surgical care was lower: 20% of the population in New England. Insurance for physician care in the hospital, office, and home had only been marketed for 2 years; consequently, only 150,000 New Englanders had it. As for consumers' enthusiasm for public insurance, Locke cautioned: "Many of these people [consumers] have clearly indicated that they want something for nothing. It is impossible to design a sound insurance plan that will give people something for nothing. The only way to give people something for nothing is through the tax system, although overall the taxes must be paid by the people."

Twenty Five Years Ago

[May, 1974]

Pierre M. Galletti, MD, PhD, Vice President, Biology and Medicine, Brown, described a proposed "Regional Network for Maternal and Infant Care." He set the goals of this system: reduction of maternal mortality toward 1.1/10,000 live births, reduction of perinatal mortality toward 12/1,000 newborns newborns > 1,000 grams; reduction in perinatal morbidity to minimize the number of newborns suffering from obstetrical trauma or asphyxia, and simultaneous reduction of the incidence of premature births. "All of these objectives may sound ambitious. Yet ... they have been reached, or will soon be reached, in other countries."

Rudoph W. Pearson, MD, and Joseph P. Bellino, MD, in "Epistaxis Due to Esthesioneuroblastoma" urged physicians to examine patients with severe nosebleed "with a nasopharyngoscope within two weeks of discharge from hospital or office to rule out tumors of nasal cavity and nasopharynx." In 100 cases, the authors had found 3 patients with such tumors. The treatment was radiation following biopsy. Two died from this "...virulent malignant tumor which presents with the mildest of symptoms—nasal obstruction and blood-stained mucus."

An editorial on "Model Cities and Neighborhood Health Centers" noted that South Providence led the state's census tracts for low birth weight babies, infant deaths, tuberculosis, venereal disease, influenza, homicide, suicide, and cancer mortality, but cautioned that health centers would not alleviate the causes. "The establishment of a neighborhood health clinic, while desirable, treats the symptoms of a social disorder rather than the disorder itself." Further, "Neighborhood health centers should not be expected to decrease inpatient hospitalization by the residents."

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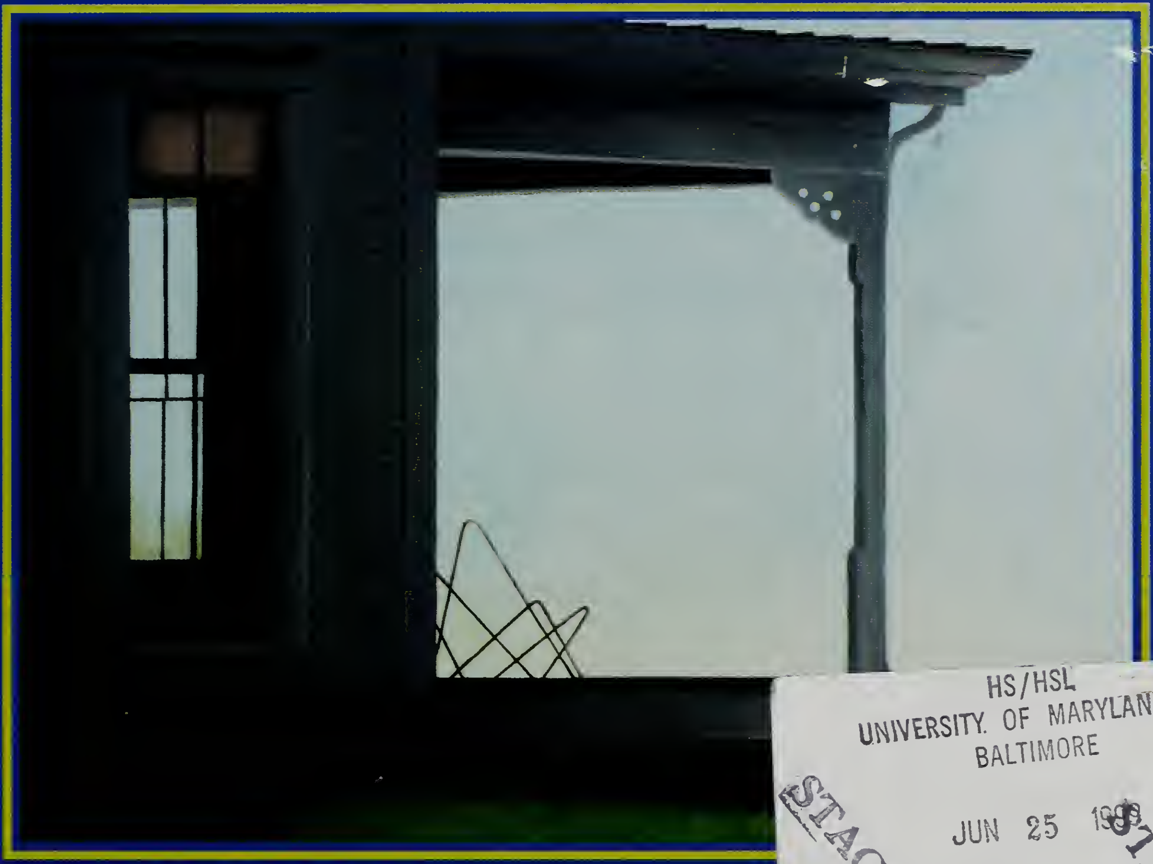

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COMMENTARIES

Active-Controlled Versus Placebo-Controlled Trials

Abbreviations Used:

FDA	Food and Drug Administration
IRB	institutional review board
NIH	National Institutes of Health
PD	Parkinson disease

It is a fundamental tenet in clinical therapeutics that the "gold standard" for demonstrating treatment efficacy is a double-blind, placebo-controlled trial. This means, of course, that the subject in the trial, always a volunteer, receives either the active treatment or a sham treatment (placebo) and neither the subject nor the investigator evaluating the treatment outcome knows which therapy the subject received until the trial is complete. The validity of this approach is well accepted for conditions that have no proven therapy. However the acceptance of this construct is less universal when there are unproven but "accepted" or time-honored therapies. An example would be heparin in acute brain infarct more than three hours old. Many physicians routinely use heparin despite knowing that no data justifies its use. Should potential anti-stroke agents be tested against placebo or heparin? The degree of acceptance of the standard therapy determines the degree of resistance to a placebo-controlled trial.

Over the past few years the concept of "sham" surgery has made the ethics of placebo-controlled trials more problematic. The *New York Times* recently brought this issue to the public's attention but clinicians have been wrestling with it for years. The study that drew the media's attention was the report on human fetal implantation for the treatment of advanced Parkinson's disease in which half the patients had a burr hole drilled and sat in the operating room while the staff mimicked a brain operation. Investigators, IRBs, ethics panels and the NIH debated this for a long time before approving the concept. It turned out that the single most impressive result of the fetal implant trial was the magnitude and duration of the placebo effect, not, unfortunately, the effect of the actual implants.

Most problematic ethically is the use of placebo-controlled trials in conditions that have known, proven treatments. Recently I was asked to participate in a double-blind, placebo-controlled trial of a drug for testing depression in Parkinson's disease patients. I thought this acceptable until I discovered that the subjects might be severely depressed and might receive placebo for six months. While it is true that no antidepressant has been proven effective in treating depression in PD patients and therefore none is approved by the FDA for this specific indication, a fair amount of data has been published indicating that antidepressants work as well in PD as in non-PD depressed people. Moreover, no other single group has been singled out for special drug approval, e.g., depression in patients with rheumatoid arthritis, or leukemia, etc. So I inquired into the issue. How can the use of a placebo be justified in "treating" severe depression (assuming mild-to-moderate depression is less disturbing and more tolerable) for six months? The surprising answer was that the FDA requires placebo-controlled trials before granting approval to any antidepressant. This is true for anti-psychotics as well, leading, of course, to major quandaries for investigators. This slows enrollment and therefore results, and has led some centers to coerce candidates unethically into studies.

The FDA's concern is statistical confidence. If one tests a new agent against an active treatment and finds no statistically significant difference this does not mean that the experimental intervention works. It is quite possible that a statistical analysis could yield a result which is not statistically different from either placebo or an effective therapy. Certainly in the area of depression, where the placebo effect is enormous, this is an important concern. However, when there are multiple proven therapies to choose from, a more ethically palatable protocol would compare a new therapy to a proven one and demand equal or better efficacy, or equal efficacy with a significantly better side effect profile, in the new agent. This approach would eliminate the senseless development of large numbers of new agents simply because of market size. It would help sway the market toward rational therapeutic outcomes and away from marketing investment.

Placebo-controlled trials are important but may have ethical drawbacks. The FDA should be in the vanguard rather the rearguard in establishing ethical standards for treatment protocols.

– Joseph H. Friedman, MD

What Hath Brazen Science Wrought?

Recent news stories describing successful cloning of mammals have prompted speculations, dire predictions and, inevitably, profound concerns that science has finally overstepped itself, arrogantly assuming liberties reserved for higher authorities. In grasping for metaphors, commentators have likened the cloning scientists to the ill-fated adventures of Icarus, Prometheus - and Dr. Victor Frankenstein.

The pinnacle of gothic horror, in the eyes of many, was embodied in the hulking, shuffling monster created by Victor Frankenstein. This creature, endowed with limitless strength but lacking a soul, had been cleverly patched together from the body remnants snatched by Frankenstein from the gallows and from nameless graves. And who might doubt that a monster from such sinister origins, brought to life not by love but by electricity, would be destined for anything but destructiveness ?

Such a story might have been written at any time but the circumstances in the year of 1816 made this a propitious moment for a moral tale such as *Frankenstein.* This was an era when science, still in its adolescence, was breaking free from its theological restraints; when the steam engine heralded limitless power; when medicine was demonstrating the rational mechanics of the human body; and when the powers of electricity, and its relationship to nervous activity, were being actively explored [and endowed by some with mystical properties.] It was a time, too, when grave-robbing for anatomical purposes was actively pursued. Truly, it was an age when each year brought startlingly new scientific revelations; but there were many sober voices now asking whether impetuous science had not gone too far, perhaps even trespassing into sacred domains. Sermons talked darkly of the Scriptural banishment of those who had eaten the fruit of a forbidden tree which provided knowledge of life and death.

Mary Wollstonecraft Godwin, author of *Frankenstein,* was the sole offspring of two remarkable parents. Her mother, Mary Wollstonecraft, was England's leading feminist; and her father, William Godwin was an eminent libertarian philosopher. Mary's life began in sorrow when her mother died of postpartem complications within days of Mary's birth. Her loveless childhood was tempered by an unresponsive stepmother and a distracted father. Yet in later years she recalled listening avidly to the eminent Dr. Erasmus Darwin, a frequent guest at the Godwin home, talk eloquently about spontaneous generation and the origins of life.

At age 17, back from a Scottish boarding school, Mary became smitten with another visitor to the Godwin salon, a young poet named Percy Bysshe Shelley. With no ties binding her to her family, Mary readily accepted Shelley's invitation to elope to the Continent despite the reality that the 23 year-old poet was already married and the father of an infant. The two crossed the Channel in July of 1814 where Mary, now pregnant, gave birth to a premature daughter who died within weeks. A second pregnancy in 1816 yielded her first son, William.

In the summer of 1816, Mary and Shelley rented a house, Maison Chapuis, on the shores of Lake Leman, Switzerland, walking distance from Villa Diodati, the temporary dwelling of Lord Byron and his physician, John Polidori. Byron and Shelley devoted their days to sailing; and in the evenings they discussed philosophic issues of mutual concern. On one summer evening [said by historians to have been stormy] Byron proposed that each write a ghost story. Byron chose to author a bit of horror concerning vampires. Mary, 18 years old, also participated; she later wrote: "I thought and pondered vainly. I felt that blank incapability of invention which is the greatest misery of authorship." But, she stated, eventually "my imagination, unbidden, possessed and guided me."

And thus this 18 year old, encumbered by an illicit liaison with Shelley, burdened by the care of her infant son, troubled by threats of suicide from Shelley's wife, and distracted by her father's many social problems, managed to compose an astonishing parable on man's perverse presumptions in exploring beyond the customary limits of research. She called her novel, *Frankenstein.*

Mary Shelley [she eventually married Shelley after the suicide of his first wife] subtitled her work, *The Modern Prometheus,* tacitly reminding the readership of an earlier attempt to thwart divine will when the titan, Prometheus, imprudently stole from the gods the gift of holy fire and bestowed it upon humankind. In punishment, Prometheus was chained to a mountain and each day for all eternity was attacked by the eagle of Zeus.

Frankenstein is an intricately woven tale within a tale of amoral strivings, of naive aspirations, of thoughtless, impulsive science, and of the poignant attempts of a nameless creature struggling to learn language and the mysterious ways of mortals. The story is substantially more complex than a *ménage à trois* involving a demented, grave-robbing scientist, a psychopathic assistant, and a stumbling, menacing Boris Karloff. The assembled creature fails to understand his origins, is eventually abandoned by his creator and then wreaks pointless vengeance on innocent souls. Both Victor Frankenstein, the striving scientist from Ingolstadt, and his creature ultimately perish in the Arctic wastes. From the start, the two were doomed by the moral constraints of 19th Century fiction-writing; but doomed, too, by a society which worshipped Creators but was dismayed by creative acts.

Mary Shelley's writing career did not stop with *Frankenstein.* In her life of 53 years she published two travel books, edited her husband's poetry, and wrote six works of fiction including *The Last Man* [1826] a novel depicting an apocalyptic plague which destroys all but one human. Writing was her salvation, even if her memories forbade the writing of joyous, romantic novels. "My dreams were all my own; I accounted for them to nobody; they were my refuge when annoyed - my dearest pleasure when free."

From birth, Mary Shelley lived in the shadows: as the murderer [so she believed] of her mother; as an outcast child; as the reviled and marginalized companion to Shelley; as an unfulfilled writer; and as one who saw three of her children die. On the title page of *Frankenstein* she quotes Milton: "Did I request thee, Maker, from my clay, To mould me man ? Did I solicit thee From darkness to promote me?" Were these the distilled thoughts of the Monster, or merely Mary contemplating her own life ?

– Stanley M. Aronson, MD

A Glimpse Into Diagnostic Radiology Today

Abbreviations Used:

CNS	central nervous system
CT	computerized tomography
MR	magnetic resonance imaging
PET	positron emission tomography
RFA	radiofrequency ablation

I am thankful to our new editor, Joseph Friedman, for asking the Department of Diagnostic Imaging at Brown University to contribute a radiologic update to *Medicine & Health/ Rhode Island.* This is an opportunity for our department to describe some of the pervasive and dramatic innovations that are occurring in the field of diagnostic imaging. It is a challenge for any single radiologist to be fully aware of the nuances in a subspecialty area as well as to have a broad working knowledge in other areas of imaging. In preparing this issue, I have drawn upon the expertise of a few subspecialty radiologists. The topics were chosen because these are areas undergoing extensive change and would impact the largest number of physicians.

There are several overriding trends in imaging. A decade ago opinion held that computerized tomography (CT) had reached its maximum capabilities, but today we realize that CT has unlimited growth potential. The development of CT spiral imaging allows the radiologist to evaluate disease in all areas of the body. The CT scanner has become an essential diagnostic tool in the emergency room. For the evaluation of abdominal pain, weight loss, and other non-specific complaints encountered so frequently by clinicians, CT is the final arbiter for the necessity of additional evaluation. Chest disease is diagnosed effectively with CT; the lung parenchyma, the pleura, the mediastinum, as well as even embolic disease, can be diagnosed with ease. The role of the non-contrast radiograph (chest x-ray) is more limited but remains a valuable screening examination which often prompts further work-up with more advanced imaging modalities. William Mayo-Smith discusses the role of CT in the evaluation of the acute abdomen. Gerry Abbott discusses the role of CT in evaluating pulmonary embolism.

The pediatric patient lacks a significant amount of intra-abdominal fat. This diminishes the utility of CT in the abdomen and the pelvis. In this patient population, ultrasound has become the dominant diagnostic tool, and Ken Mendelson, a pediatric radiologist, reviews the role of ultrasound in evaluating the pediatric patient presenting with acute abdominal pain.

No other organ system has benefited more from the growth in diagnostic imaging than the nervous system. Technical advances in MR (magnetic resonance) imaging permit us to image the brain with exquisite anatomical detail. In addition, MRI is challenging nuclear medicine because of its ability to indicate function and viability of brain tissue. These imaging capabilities have already begun to change treatment options. Similarly, interventional neuroradiology has become almost a mundane practice in a busy specialty setting, but it is relatively unknown to the physicians practicing outside of the neurosciences. In this section, Glenn Tung, a neuroradiologist, and his colleagues explain some of the current advances in neuroimaging and neurointerventional radiology.

Nuclear medicine is often the forgotten stepchild of diagnostic imaging. For decades soothsayers have predicted the demise of nuclear medicine based on the development of CT, ultrasound, and MRI. In reality, nuclear medicine remains strong and viable because of new techniques, which are dependent upon improved gamma cameras, computers, and new isotopes. The ability to display functional imaging vs. morphology remains nuclear medicine's strength. While there are no positron emission tomography (PET) scanners in the Rhode Island area, a new technique, coincidence scanning with a modified gamma camera, is now operational in the state. This procedure allows functional assessment of metabolism in the CNS as well as determination of subtle tumors in the head, chest and heart, similar to the diagnostic information provided by PET. Richard Noto and Jac Scheiner bring us up-to-date in these developments.

Interventional techniques combined with cross-sectional imaging permits minimally invasive procedures to be performed. Percutaneous biopsy and abscess drainage techniques have been firmly established in the therapeutic armamentarium. For over a decade we have attempted to control metastatic or focal primary disease with percutaneous alcohol injections or catheter embolizaitons. Damian Dupuy provides a unique perspective on Brown's position as a world leader in the application of radiofrequency ablation (RFA) to treat tumors. Initially, RFA was limited to the treatment of liver lesions or small benign bone lesions. However, we have now treated lung lesions, renal lesions, head and neck malignancies and almost any other process that is accessible to the electrode. I trust that your reading of this short article will be of great use.

In summary, this edition of *Medicine & Health/Rhode Island* provides an opportunity to review some of the newer techniques in radiology. Change continues, and I am confident that we could do another radiologic update in a very short period of time.

– John J. Cronan, MD

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Information for Contributors

Medicine & Health/Rhode Island welcomes submissions from members of the Rhode Island health care community. Submissions can fall into one of three categories:

CONTRIBUTIONS

Contributions should report on an issue of interest to clinicians in the state: new research, treatment options, collaborative interventions, review of controversies. The maximum length of submissions is 2500 words; the maximum number of footnotes is 15. (The Journal is not the venue for an exhaustive literature review). Tables, charts, and figures should be camera-ready. Photographs should be black and white. (Slides are not accepted).

Creative Clinician

Clinicians are encouraged to submit brief (no more than 1200 words) descriptions of cases that defy texbook analysis. Photographs, charts and figures may accompany the case; footnotes should not exceed 6.

POINT OF VIEW

The column gives readers an opportunity to share their perspective on any issue facing clinicians. The topic is broad: it could, for example, include ethics, health care policy, and/or relationships with patients. Maximum length is 1200 words.

The format of submissions is as follows:

The title page should include name, affiliation, address, phone, fax, and e-mail. A brief abstract should appear on a separate page. References should be numbered sequentially in the text, and listed separately at the end of the document (not the end of each page).

For Contributions and Point of View, please submit 4 hard copies of the document, with a disk (Microsoft Word or Text), to the managing editor: Joan Retsinas, PhD, 344 Taber Avenue, Providence, RI 02906.

For Creative Clinician columns, please submit 3 hard copies of the document, with a disk (Microsoft Word or Text), to Anthony Mega, MD, Miriam Hospital, Providence, RI 02906.

For additional information, please contact Joan Retsinas (phone: (401) 272-0422; fax: (401) 272-4946; e-mail: JRetsinas@aol.com).

BOOK REVIEWS

Medicine & Health/Rhode Island will review books authored by Rhode Island physicians. Publishers should send a copy for review to the managing editor.

Advances in Nuclear Medicine: Current Concepts

Richard B. Noto, MD, and Jac D. Scheiner, MD

The field of nuclear medicine has evolved rapidly in recent years with new procedures and new radiopharmaceuticals developing to visualize a wide variety of disease processes. In the 1970s and early 1980s, the most common scintigraphic procedures included studies such as liver-spleen and conventional brain scans which have almost entirely disappeared from clinical use. These procedures have been replaced with a variety of studies which take advantage of the unique functional information nuclear medicine can provide. Recent advances include the use of improved blood flow tracers for cardiac and brain studies in conjunction with SPECT (single photon emission computed tomography) imaging, the evolution of several agents for tumor imaging including radiolabeled peptides and monoclonal antibodies, and the improved availability of F-18-fluorodeoxyglucose (FDG) coincidence detection imaging.

Abbreviations Used:

CABG	coronary artery bypass graft
ECD	ethyl cysteinate dimer
FDG	fluorodeoxyglucose
HMPAO	hexamethylpropylenamine oxime
MCD	multiple coincidence detection
PTCA	percutaneous transluminal coronary angioplasty
PET	positron emission tomography
SPECT	single photon emission computed tomography

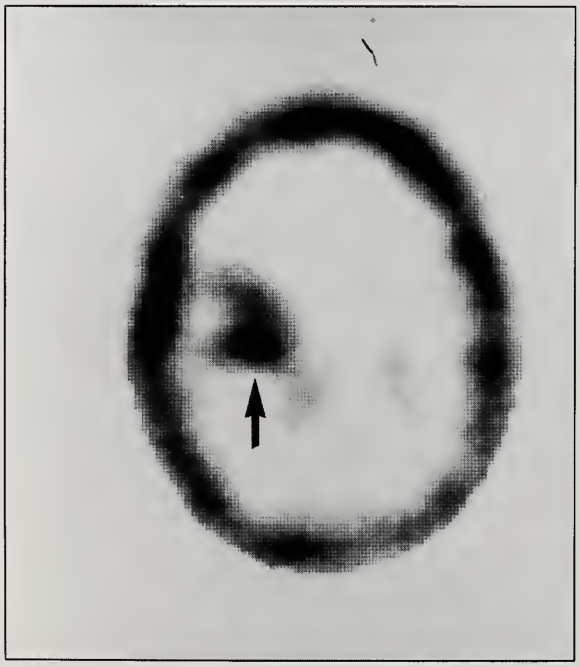

Fig. 1 Axial SPECT thallium 201 image of the brain demonstrates abnormal uptake in the right front-parietal region (arrow) consistent with tumor recurrence.

BRAIN SCINTIGRAPHY

Nuclear medicine evaluation of the brain has evolved dramatically due to the availability of brain perfusion agents and improvements in brain SPECT imaging quality. Two radiolabeled lipophilic amines, technetium 99m hexamethylpropylenamine oxime (HMPAO) and technetium 99m ethyl cysteinate dimer (ECD), cross the blood-brain barrier and localize in the brain in proportion to regional cerebral blood flow. The technical quality of brain SPECT scans has improved greatly with the evolution of multiple head gamma cameras and dedicated ring-detector brain SPECT cameras. This procedure has found its greatest utility in imaging patients with dementia where a brain SPECT can differentiate Alzheimer's disease from other types of dementia in the majority of cases. Alzheimer's disease most commonly shows a pattern of diffuse bilateral temporoparietal hypoperfusion which can usually be distinguished from vascular causes of dementia, frontal dementias such as Pick's disease, and pseudodementia of depression.[1] Sensitivity for Alzheimer's disease improves with increased disease severity, but is at least 80% even in milder cases.[2,3]

Other potential uses for brain SPECT include localization of seizure foci in epileptic patients, detection of acute ischemic events, evaluation of AIDS encephalopathy, and visualization of sequelae of head trauma.[2] In addition, brain SPECT with thallium 201 is highly accurate in differentiating radiation necrosis from residual or recurrent tumor in patients who have received radiation therapy for brain tumors.[4] (Figure 1)

MYOCARDIAL PERFUSION SCINTIGRAPHY

Myocardial perfusion scintigraphy continues to be the imaging modality of choice in many patients with suspected or known coronary artery disease. Indications for myocardial perfusion scans include detection of previously undiagnosed coronary artery disease, evaluation of the physiologic significance of known coronary artery disease, determination of the presence and extent of stress-induced ischemia in patients with recent myocardial infarction, risk stratification before high-risk non-cardiac surgery, and assessment of restenosis after percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass graft (CABG).[5]

In addition to thallium 201, which has been used for many years, technetium labeled agents such as technetium 99m sestamibi and technetium 99m tetrofosmin have become available and have many potential advantages. The technetium agents offer superior penetration of overlying soft tissues, minimizing soft tissue artifacts. In addition, the higher count rates available with the technetium radiopharmaceuticals enable the acquisition of ventricular function data, either through first pass acquisition or, more commonly, through gated SPECT wall motion analysis. Gated SPECT imaging is useful in evaluating the overall left ventricular function and can provide an accurate estimate of ejection fraction. In addition, gated SPECT can allow evaluation of regional wall motion which can be extremely helpful in separating attenuation artifacts from real fixed defects due to coronary artery disease.[6] Attenuation correction programs have also been devised which help to minimize artifacts from overlying soft tissue.

Evaluation of myocardial viability is often possible with myocardial perfusion SPECT, but single photon tracers often underestimate the extent of underperfused but viable myocardium. Imaging with FDG positron emission tomography (PET) is a superior technique for identifying viable myocardium but up to this time has not been readily available because of the expense of the PET scanner and need for a cyclotron to produce FDG. However, recent advances in nuclear medicine camera technology now allow modified gamma cameras to perform coincidence detection imaging of positron emitters and FDG has become available from central radiopharmaceutical distributors, eliminating the need for an on-site cyclotron. With these changes, FDG imaging will be much more widely available and this may change the methods for evaluating myocardial viability in the near future.

ONCOLOGIC IMAGING

Nuclear medicine has long played a role in imaging of oncologic patients. Procedures such as bone scintigraphy for evaluation of bone metastases and radioiodine imaging for detection of functioning thyroid metastases remain the standard of care. However, a variety of new scintigraphic techniques have become available for the diagnosis of different tumors and this area continues to evolve more rapidly than any other in nuclear medicine.

Among the new radiopharmaceuticals that have received FDA approval since 1992 are both radiolabeled monoclonal antibodies and radiolabeled peptides for receptor imaging. A large number of monoclonal antibodies have been studied over the years with mixed success. Early expectations for radioimmuno-scintigraphy to become a "magic bullet" for imaging of tumors have not been fulfilled, but four radiolabeled monoclonal antibodies are currently approved for tumor imaging and play a useful role in specific situations. The four currently available monoclonal antibodies, in order of FDA approval, are: 1) Indium-111 satumomab pendetide (OncoScint CR/OV) for colorectal and ovarian carcinoma imaging; 2) Technetium-99m arcitumomab (CEA-Scan) for colorectal carcinoma; 3) Technetium-99m nofetumomab merpentan (Verluma) for small cell lung cancer; and 4) Indium-111 capromab pendetide (ProstaScint) for prostate carcinoma.[7] The most common applications of these agents have been as an adjunct to conventional modalities in tumor staging and in evaluating for possible recurrent disease in patients with rising tumor markers but negative or equivocal conventional imaging studies.

Perhaps even more promising than antibody imaging is the field of receptor imaging. Receptor ligands are small peptides which are much smaller than intact antibodies.[8] They bind to specific cell surface receptors and often provide superior contrast than monoclonal antibodies. The first of these agents, Indium-111 pentetreotide (Octreoscan), was approved for routine use in 1994; this somatostatin analog is useful for tumors which have somatostatin receptors. The most common utilization of this agent is for localization of functioning neuroendocrine tumors of the gastrointestinal tract and pancreas, such as carcinoids and pancreatic islet cell tumors. Indium-111 pentetreotide also has high sensitivity for pheochromocytoma (Figure 2) and small cell lung carcinoma. A number of other receptor agents are currently under development which have great potential in imaging a variety of different tumors.

Another area in which nuclear medicine is making an increased contribution is breast cancer. Technetium-99m sestamibi, the same radiopharmaceutical used for myocardial perfusion imaging, has been shown to have high sensitivity and specificity for detection of breast cancer, especially those lesions that are greater than 1.0

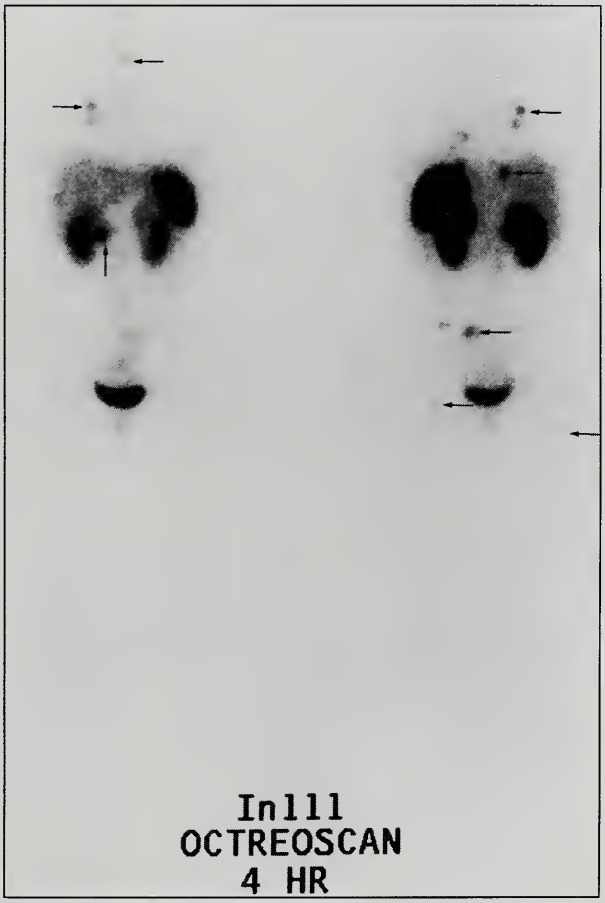

Fig. 2 Anterior and posterior whole body images obtained four hours after administration of 5 mCi of In-111 pentetreotide. Multiple metastases (arrows on some of the tumors) are demonstrated in the thorax, abdomen, pelvis and lower extremities in a 35-year-old woman with malignant pheochromocytoma.

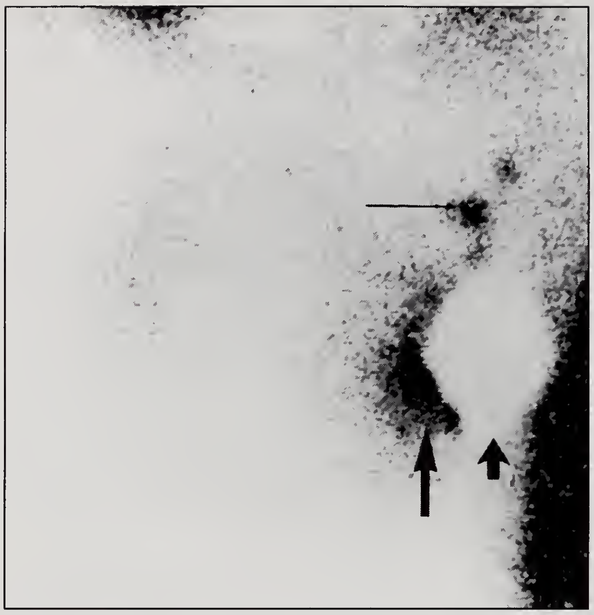

Fig. 3 Anterior image of the chest obtained after intradermal injection of technetium-99m sulfur colloid about site of known left breast cancer. The sentinel node (arrow) is visualized in the left axilla. The round area of decreased uptake (short thick arrow) corresponds to a lead blocker which partially covers the injection site (long thick arrow).

cm. in size.[9] Unlike mammography, mammoscintigraphy with sestamibi is not affected by the overall density of the breast parenchyma and may also show lymph node metastases. While mammography clearly remains the screening study of choice, current potential indications for sestamibi breast imaging include evaluation of high-risk patients with dense or post-surgical breasts, poorly defined palpable abnormalities with negative mammography and/or ultrasound, patients with suspected multifocal disease, and evaluation of therapeutic response.[10]

More recently, breast lymphoscintigraphy has become an essential technique in the work-up of patients with known breast cancer. Lymphoscintigraphy, performed with technetium-99m sulfur colloid administered near the site of the breast tumor, is capable of localizing the sentinel axillary lymph node with a high degree of accuracy. The sentinel node is the main site of lymphatic drainage which is predictive of overall axillary lymph node status in greater than 95% of cases.[11] During lymphoscintigraphy, the sentinel node is first detected by gamma camera imaging (Figure 3) and later localized in the operating room with a hand-held gamma probe. Breast lymphoscintigraphy can markedly reduce the number of patients who require a more extensive axillary dissection and often limits the surgery to a single sentinel node without losing diagnostic accuracy in axillary lymph node staging.

It has become increasingly apparent in recent years that nuclear medicine imaging with gallium provides critical information in the work-up of patients with lymphoma. At initial patient presentation, whole body and SPECT gallium scans provide highly accurate staging information regarding disease extent. Specifically, these scans can detect lymphoma sites which are not visualized by conventional CT scanners. In addition, they can discern the oncologic significance of smaller nodes seen on CT, as well as the significance of a post-therapy residual mass (residual tumor vs. scar tissue).

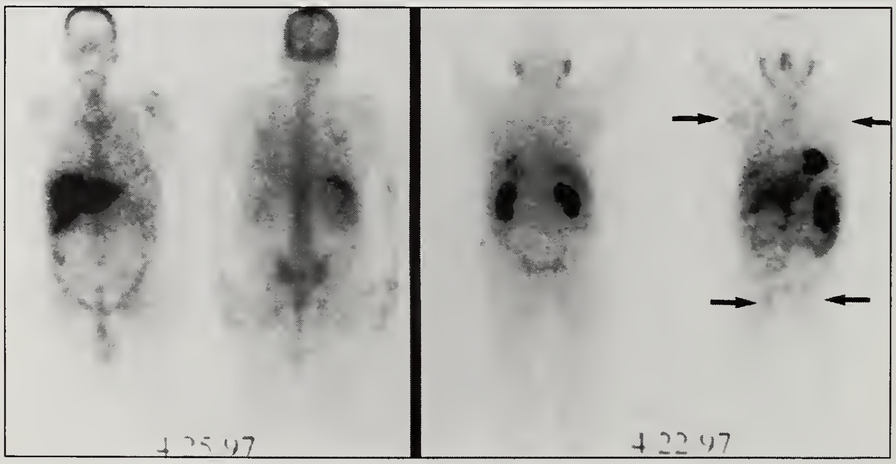

Fig. 4 Anterior and posterior whole body images were obtained using gallium (4/25/97) and thallium (4/22/97). Lymph nodes in the inguinal and axillary regions were only seen on thallium images in this patient with low grade lymphoma (arrows).

It is important to note, however, that gallium uptake by lymphoma is proportional to tumor grade. The sensitivity of gallium in the detection of low grade lymphoma is decreased. Fortunately, it has recently been demonstrated that thallium-201, which is also used in cardiac imaging, is highly accurate in the evaluation of low grade lymphomas. (Figure 4) A study at Cedars-Sinai hospital in Los Angeles demonstrated that approximately 44% of sites of low grade lymphoma were only seen on thallium scans, and not on gallium scans.[12] Conversely, all sites of low grade lymphoma seen on gallium scans were seen on thallium scans. Both scans can play important roles in the management of patients with low grade lymphomas. The thallium scan detects the sights of low grade lymphomas, whereas the gallium scan detects sights that have converted to a higher grade.

MULTIPLE COINCIDENCE DETECTION (MCD) IMAGING

Radiology has long been a dynamic and exciting field. The 1970s and 1980s brought the advent of computed tomography (CT) and magnetic resonance imaging (MRI). These imaging modalities provided exquisite anatomic detail to aid in disease diagnosis. In the 1990s, another diagnostic imaging modality has come into the forefront, emphasizing disease physiology (as opposed to morphology): the imaging of radioactive glucose (FDG) using positron emission technology.

CT and MRI are excellent at what they are designed to do: detect anatomic abnormalities. However, these modalities have limitations. For example, these modalities may demon-

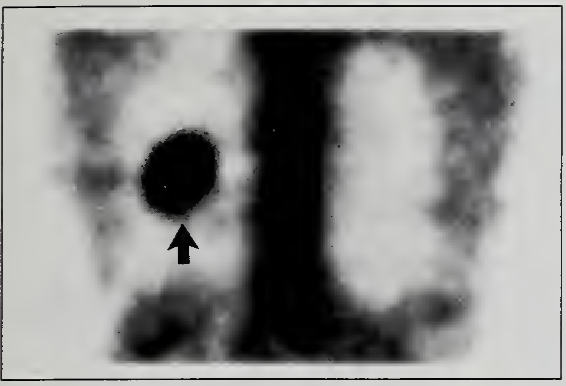

Fig. 5 Coronal reconstruction from an FDG coincidence detection study with attenuation correction. Increased FDG uptake is noted in a right lung cancer (arrow) in a 69-year-old male. (Image courtesy of ADAC laboratories.)

strate a solitary pulmonary nodule. While the nodule may represent a benign lesion (such as a granuloma), it may also represent a subtle lung cancer. FDG imaging provides a method to distinguish these entities. Metabolically active lesions, such as lung cancer, tend to take up FDG (Figure 5) whereas benign lesions rarely show significant FDG uptake.

The gold standard for FDG imaging is the use of a specially designed camera, know as a Positron Emission Tomography (PET) camera. The sensitivity and specificity of PET for distinguishing benign from malignant pulmonary nodules is quoted to be approximately 96% and 88%, respectively.[13] However, due to the expense, a minority of hospitals have access to these cameras. Fortunately, less expensive cameras have recently been developed (called MCD cameras), which can image FDG with similar results. A recent study from the University of Michigan showed that FDG imaging using MCD detected 93% of the cancerous solitary pulmonary nodules detected with PET.[14] Recent technological improvements, such as attenuation correction, should improve these results.

Another application of MCD imaging will be in the pre-operative staging of lung cancer. The finding of contralateral lymphadenopathy in lung cancer patients renders the tumor nonresectable. CT is the current screening tool used to detect these lymph nodes. However, lymph node size is not always an accurate predictor of malignancy and if the lymph nodes seen on CT are 1 cm or less in diameter, it is unclear whether these nodes are due to metastatic disease or benign inflammation. MCD imaging plays a vital role in these cases, since intense uptake of FDG by the lymph nodes is highly predictive of metastatic disease. This information may save lung cancer patients unnecessary morbidity related to non-indicated pneumonectomy. Other oncologic applications of MCD imaging include melanoma staging, as well as distinguishing post-operative changes from tumor recurrence in patients with brain neoplasms. Utilization of FDG imaging in a number of other tumors is currently under intensive investigation. Non-oncologic applications of MCD imaging include myocardial viability assessment and cerebral seizure focus localization.

Recent advances include the use of improved blood flow tracers for cardiac and brain studies in conjunction with SPECT (single photon emission computed tomography) imaging, the evolution of several agents for tumor imaging including radiolabeled peptides and monoclonal antibodies, and the improved availability of F-18-fluorodeoxyglucose (FDG) coincidence detection imaging.

SUMMARY

As described above, many scintigraphic studies have recently become available which provide vital clinical information in a wide variety of diseases. The future of nuclear medicine also appears bright.[15,16] Modifications to current gamma cameras will likely occur in the next few years to replace sodium iodide crystals with new scintillators like oxyorthosilicates which have better imaging characteristics for both single photon and positron-emitting radionuclides. Ongoing computer improvements will allow improved reconstruction and processing with newer systems already capable of advanced iterative reconstruction algorithms. Development of new radiophamaceuticals is progressing at a rapid pace, with emphasis on new tracers that can provide information about diseases at the molecular level, such as receptor imaging agents. In addition, radiolabeled tracers for therapeutic purposes will continue to progress, including monoclonal antibodies labeled with beta emitters. Overall, nuclear medicine will continue to evolve in many new directions and provide useful functional imaging data about a variety of different organ systems and diseases.

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Evaluation of Pulmonary Emboli: Current Concepts

Gerald F. Abbott, MD

Acute pulmonary embolism (PE) is an important cause of mortality, particularly in hospitalized patients. It is estimated that over 600,000 symptomatic episodes of PE occur per year in the United States, causing approximately 200,000 deaths.[1] In spite of this incidence, the clinical diagnosis of PE remains challenging and sometimes elusive. In recent decades, various imaging modalities have been employed to meet this challenge. Each has improved our diagnostic abilities, but no single test has proven to be the answer.

The recent introduction of spiral CT has modified the diagnostic work-up of pulmonary embolism by noninvasively depicting endoluminal clots in pulmonary arteries. This paper will review the various imaging modalities currently used for the evaluation of pulmonary emboli and the present role of spiral CT in the work-up of these patients.

Abbreviations Used:

CT	computed tomography
DVT	deep vein thrombosis
PE	pulmonary embolism
PIOPED	Prospective Investigation of Pulmonary Embolism Diagnosis
US	ultrasound
V/Q	ventilation-perfusion

IMAGING MODALITIES

Chest radiography:

In most instances, pulmonary emboli do not produce abnormalities on the chest radiograph. In fact, the diagnosis of acute PE should be considered in a patient with acute respiratory distress and a normal chest radiograph. When abnormalities are related to pulmonary embolism, the radiograph may demonstrate volume loss, pleural effusion, consolidation (Hampton's hump), increase in hilar vessel size with abrupt tapering (knuckle sign), or oligemia (Westermark's sign). In addition, the chest radiograph is useful to a) detect other thoracic abnormalities that may mimic PE and, b) assist in the interpretation of ventilation-perfusion scans.[2]

Radionuclide (V/Q) scanning:

Radionuclide lung ventilation-perfusion (V/Q) scanning is the standard initial pulmonary imaging test in suspected pulmonary emboli. It is performed by administering an intravenously injected perfusion agent (technetium 99m-macroaggregated human albumin) and an inhaled ventilation agent (usually Xenon-133). V/Q scanning does not permit direct visualization of a PE. Rather, it relies on indirect evidence: a ventilation-perfusion mismatch between the perfusion and ventilation portions of the V/Q

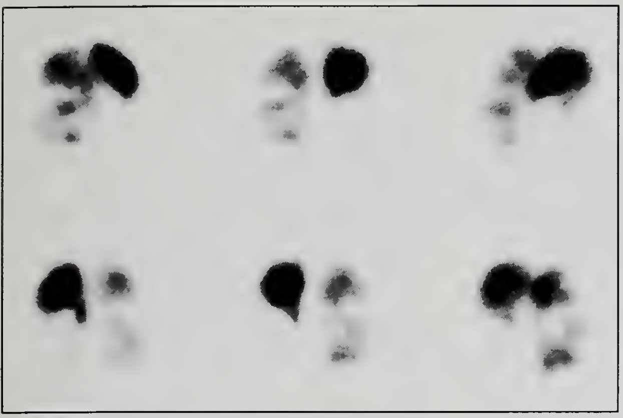

Figure 1. Technetium-99m-MAA perfusion study in a 62-year old female with multiple pulmonary emboli. Multiple views demonstrate bilateral segmental and subsegmental wedge-shaped pleural based perfusion defects. There were no corresponding ventilation defects ("matches") on the concurrent Xenon-133 ventilation study. This pattern fits the high probability diagnostic category using PIOPED criteria.

study.[3]

The criteria for interpreting V/Q scans were established by a large prospective multi-institutional investigation, the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study. Using those criteria, V/Q scans are categorized as 1) normal, 2) low probability for PE, 3) intermediate probability for PE, or 4) high probability for PE. (Figure 1) The original criteria were recently modified to allow better discrimination between intermediate and low probability V/Q scans.[4,5]

When the scan is interpreted as normal or as indicating a high-probability of PE, the clincal question is usually answered satisfactorily. A low-probability interpretation may also be satisfactory, particularly if the clinical suspicion of PE is low. All scans which cannot be categorized as high probability, low probability or normal are, by definition, intermediate category studies.[3,6]

When the V/Q scan is interpreted as intermediate and clinical suspicion of PE remains high, additional imaging studies are warranted. While pulmonary angiography remains the "gold standard" in the diagnosis of PE, non-invasive studies such as compression ultrasound (US) of the lower extremities and spiral CT angiography of the pulmonary arteries have established themselves as useful complementary imaging modalities.

When interpreting V/Q scans, it is important to correlate the findings with a current chest radiograph to exclude other causes of a V/Q mismatch (neoplasm, vasculitis, pulmonary artery stenosis or atresia).

Ultrasound:

Based on the fact that the majority of PEs originate in lower extremity veins, compression US of the lower extremities has been established as a dependable and widely available technique for the diagnosis of deep vein thrombosis (DVT). Clinicians may request bilateral lower-extremity US studies when suspicion of PE exists and, if the results are positive, may rationally begin anticoagulation therapy. Even in the setting of an indeterminate V/Q scan, it has been shown that the chance of PE is greater than 90% when lower-extremity DVT is documented by US.[7]

It must be noted, however, that less than one-half of patients with angiographically-proven PE have US evidence of DVT. Therefore, negative findings of a lower extremity US study do not exclude the possibility of PE.[8,9]

Pulmonary angiography:

Pulmonary angiography is currently regarded as the most accurate imaging test for diagnosing acute PE ("the gold standard"). While pulmonary angiography is invasive and not without risks, the complication rate of the procedure is relatively low, estimated at 1-5%, while the mortality is approximately 0.2%. The mortality increases to approximately 2% in patients with pulmonary hypertension. Pulmonary emboli manifest as intravascular filling defects with associated abrupt vascular cut-off on pulmonary angiographic studies. (Figure 2) It is generally accepted that a negative pulmonary angiogram is sufficient to exclude acute PE.[2]

Spiral (helical) CT:

In recent years, contrast-enhanced spiral CT has been established as a useful modality for detecting pulmonary emboli.[10] Spiral, or helical, CT scan-

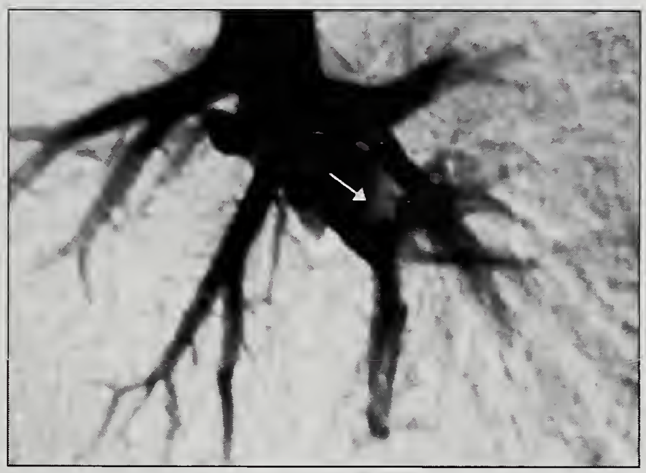

Figure 2. Pulmonary arteriogram in a 71-year old male with left lower lobe pulmonary embolus. Injection of the left pulmonary artery reveals clot in a left lower lobe segmental artery (arrow).

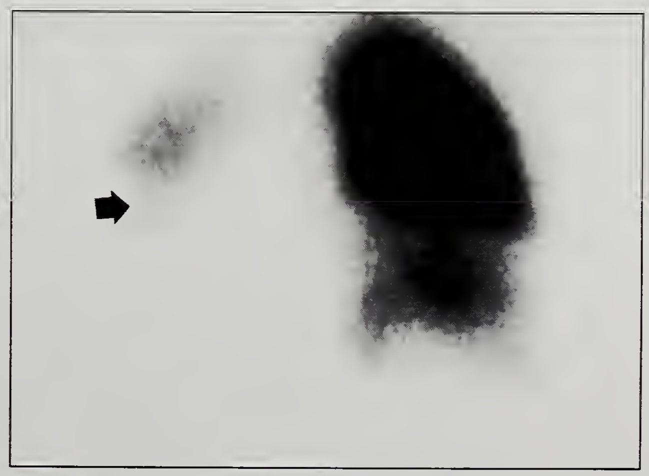

Figure 3a. Pulmonary embolism in a 54-year old male with shortness of breath and a high-probability V/Q scan. Technetium-99m-MAA perfusion study (posterior view) demonstrates marked decreased perfusion of the left lung (arrow).

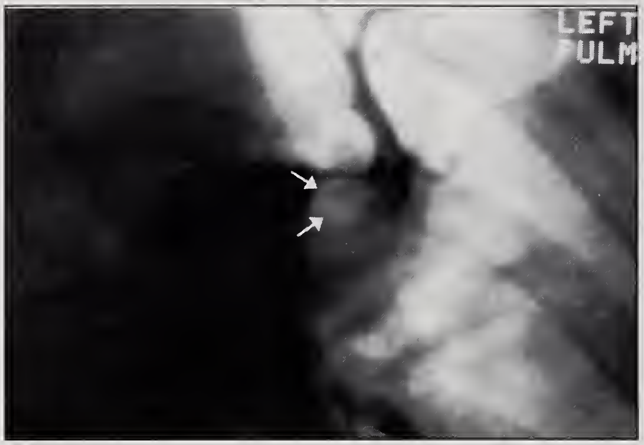

Figure 3b. Pulmonary arteriogram with injection of the left pulmonary artery demonstrates clot almost totally occluding the arterial lumen (arrows).

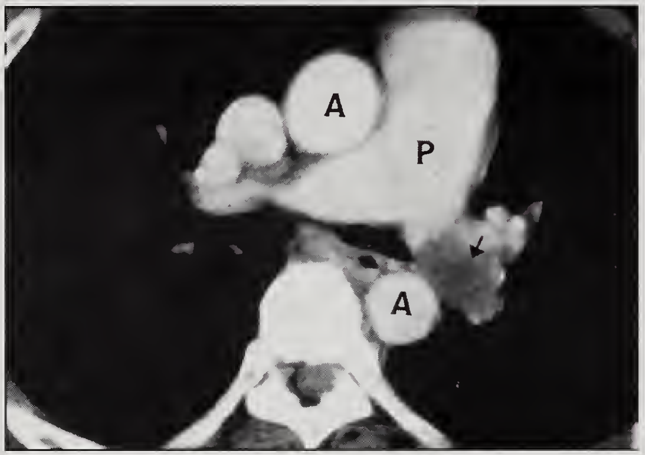

Figure 3c. Contrast-enhanced spiral CT scan shows endoluminal filling clot (arrow) in the left pulmonary artery which corresponds to the finding on the pulmonary angiogram (see 3b). (P = main pulmonary artery, A = aorta)

ning represents a significant advance in medical imaging techology, enabling a larger volume of tissue to be imaged during a single breath-hold and resulting in improved scanning rates, excellent contrast enhancement, reduction in partial-volume effects, and reduced respiratory misregistration.[11]

In conventional CT scanning, each individual image ("slice") is obtained during a patient's single breath-hold. Ideally, each image represents a volume of tissue that is exactly contiguous with the tissue represented in the preceding and proceeding images. Unfortunately, variations in patient respiratory efforts may cause significant variations in the sequential registration of consecutive images, causing gaps in the imaging data and degrading the resolution of the study.

By comparison, spiral CT employs a technique for volumetric imaging, avoiding such gaps in data and thus improving resolution. During spiral CT scanning, the x-ray beam source and its coupled array of detectors (aligned on opposite sides of the patient) rotate continuously while the table slowly moves the patient through the scanning beam during a single breath-hold. This results in a spiral-shaped volume of scanning data which can be manipulated and displayed as individual images (reconstructed "slices") or as 3-D reconstructed images to optimize visualization of desired structures.[11]

Excellent contrast enhancement is obtained more consistently with spiral technique than with conventional CT. Spiral CT data acquisition typically begins approximately 20 seconds after the start of the intravenous contrast injection, ideally administered with a power injector. Patients are instructed and rehearsed to perform deep inspiration as scanning commences. Many patients can successfully hold their breaths for 24-32 seconds; others may require modifications in the spiral CT scanning protocol.[11,12]

The advantages of spiral CT are primarily related to its ability to obtain a uniform and high degree of arterial enhancement of pulmonary arteries, allowing direct visualization of clot within pulmonary arteries. (Figure 3) In some studies, pulmonary arteries as small as 2-3 mm in diameter have been visualized. To achieve this goal requires experience in spiral CT angiography, as well as familiarity with techniques of data acquisition and contrast medium injection.[10,12] Pitfalls in the interpretation of spiral CT images have been recognized, but these are also less problematic for radiologists with experience in spiral CT of the pulmonary vasculature.[13]

In most instances, pulmonary emboli do not produce abnormalities on the chest radiograph.

Several studies of spiral CT in the diagnosis of PE have shown an average sensitivity and specificity of 84% and 96%, prompting some to replace radionuclide scanning or conventional angiography with spiral CT.[14] The greatest sensitivity and specificity were achieved when PE involve the main, lobar or segmental pulmonary arteries. Most observers agree that the specificity of spiral CT for PE decreases significantly when clot involves pulmonary arteries at the subsegmental level or beyond.[15,16]

A major advantage of spiral CT over V/Q scanning is its ability to evaluate the morphology of the mediastinum, bronchial tree, pleura, and lung parenchyma. In one study, 11% of patients had unequivocal abnormalities of lung parenchyma on spiral CT scans that explained both the clinical presentation and the nondiagnostic results of V/Q scanning.[17]

SUMMARY

While radionuclide lung V/Q scanning remains the standard initial pulmonary imaging test in suspected pulmonary emboli, other modalities may be useful, particularly in those patients with intermediate probability V/Q scans. Compression US of the lower extremities, spiral CT angiography, and conventional pulmonary anteriography may help to establish the diagnosis of pulmonary embolism.

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Emergency Abdominal Ultrasound in Children: Current Concepts

Kenneth L. Mendelson, MD

Abbreviations Used:

CT	computed tomography
HPS	hypertrophic pyloric stenosis
KUB	non-contrast radiographs
MRI	magnetic resonance imaging
US	ultrasonography

Ultrasonography (US) has become an essential component in the evaluation of pediatric abdominal disease. The modality has several aspects that make it particularly well-suited to this age group. Most importantly, the radiologist can acquire diagnostic information without the use of ionizing radiation. Radiation dose should always be a consideration when performing diagnostic procedures on any patient, but clearly is a prime consideration in the pediatric population.

US, unlike computed tomography (CT), can be performed in almost any imaging plane. This is especially advantageous when evaluating structures such as the appendix, the pylorus, or other organs which have an inconsistent orientation. In adult US examinations, patients will usually lie quietly and suspend respiration on request. A small child with abdominal pain is less likely to do the same. Unlike CT or magnetic resonance imaging (MRI) which often require sedation (with its added expense and morbidity), ultrasound is able to obtain adequate images in non-sedated children.

The physics of US is optimal for the pediatric patient. As an US beam passes through tissue, it loses most of its energy. To interrogate the organ of interest and get back to the transducer, the beam travels a shorter distance in a child than in an adult, retaining more of the beams original energy. Because of this shorter depth, the sonographer can choose a higher frequency transducer which has inherently better resolution. The paucity of intraabdominal fat, which scatters the US beam, enhances the quality of the images in children. US can be performed at the bedside, a significant advantage over CT or MRI in the unstable patient of any age. Finally US is usually far less expensive than either CT or MRI.

The use of US in the workup of genitourinary and biliary tract complaints is well known, but US has also become the primary modality to evaluate many etiologies of vomiting and abdominal pain. This paper will discuss some of the applications of US for pediatric gastrointestinal complaints encountered in the acute setting.

Hypertrophic Pyloric Stenosis

Hypertrophic pyloric stenosis (HPS) is the most common surgical disorder producing emesis in infancy with an incidence of approximately 1 in 300 live births in the United States.[1] The classic patient is a four- to six-week-old with non-bilious vomiting. Because of the pressures generated by the hypertrophied gastric muscles, the emesis can often be quite forceful. The etiology of this condition is unclear, but there is a male predominance.

The clinical diagnosis is often straightforward. If an "olive," i.e. the mass of hypertrophic muscle, is palpated in the right upper quadrant in the appropriate clinical setting, the diagnosis is established. Recent publications have questioned the necessity and cost-effectiveness of imaging in HPS,[2,3] but the trend has been toward increased reliance on US.[4] This has resulted in a referral pattern in our practice as well as in most others from pediatrician to radiologist rather than to surgeon.

US has replaced the upper GI in the diagnosis of HPS. US avoids radiation and allows direct visualization of the pyloric muscle, as opposed to the upper GI where the morphology of the muscle is inferred by the thinness and length of the barium stream

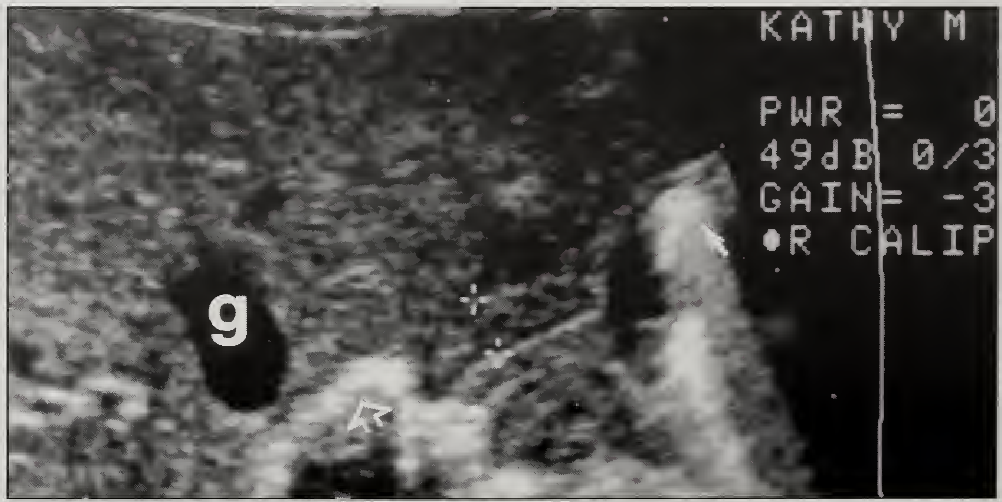

Figure 1: Transverse sonogram of the right upper quadrant demonstrating hypertrophic pyloric stenosis. Note the echogenic (white area) air in the antrum of the stomach (arrow) and duodenal bulb (arrow). The calipers are delineating one wall of the pylorus which measures 4.1 mm. The fluid-filled structure is the gall bladder (g).

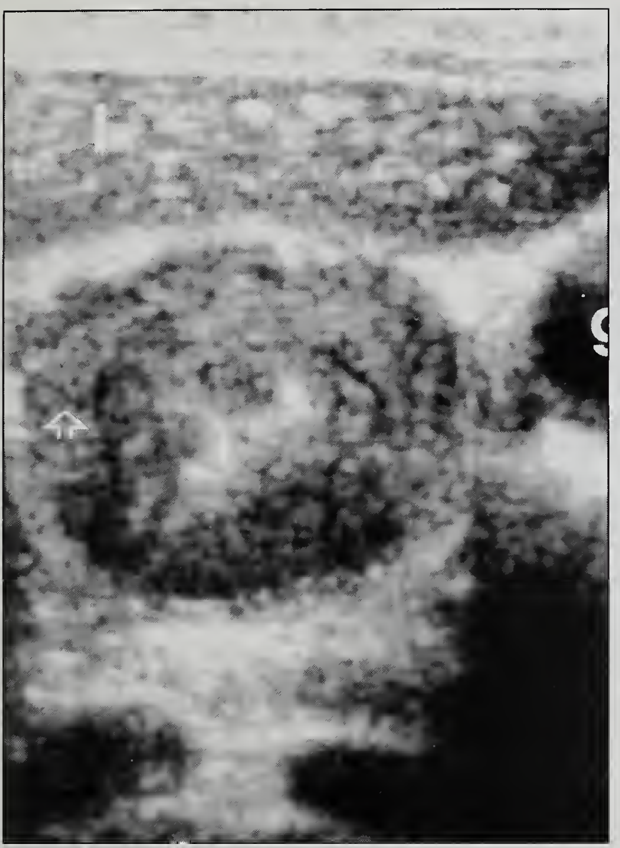

Figure 2: Transverse sonogram in the subhepatic region demonstrates an intussusception. Note the liver edge (l) and gall bladder (g). The echogenic area in the middle (arrow) is mesenteric fat in the intussusceptum (inside loop of bowel); the outer layer of the "bull's eye" is the intussuscipiens (arrow).

through the area.

The most reliable measurement of the pylorus is that of muscle thickness. In a normal infant, the thickness of a wall of the pylorus should be less than 2 mm. In HPS, the thickness measures greater than 3 mm.[5] (Figure 1) Measurements of the total diameter can lead to false positive diagnoses because of mucosal rather than muscle hypertrophy. Likewise, measurement of the length, another described parameter, can lead to false negative diagnoses if the course of the pylorus makes imaging the entire length difficult. The accuracy of US in the diagnosis of HPS approaches 100%.[5]

INTUSSUSCEPTION

Intussusception is most commonly seen between the ages of three months and four years. Approximately 65% of presenting children will be under one year of age.[6] Symptoms related to intussusception include crampy abdominal pain, vomiting, bloody (or "currant jelly") stools, and sometimes a palpable mass. Lethargy can also be seen, often as a harbinger of toxicity.

Non-contrast radiographs are often the first studies ordered. Findings are variable and depend on the duration of symptoms and presence of complications.[7] Almost all cases of idiopathic intussusception are ileocolic. In some cases, the head of the intussusceptum (the inner portion) can be seen outlined by air in the colon, often in the subhepatic region. Other times a small bowel obstruction is present along with a paucity of gas on the right. Often radiographs are normal, though a left lateral decubitus view can sometimes demonstrate air filling the cecum and ascending colon, making the diagnosis unlikely.

A contrast enema has been the traditional method to diagnose intussusception, but US has begun to supplant it as a screening modality. US has been shown to have a sensitivity of 100% with a specificity of 88%.[8] High frequency transducers can delineate the layers of the intussusception, demonstrating a round or oval mass with both sonolucent (muscle) and echogenic (mesenteric fat) stripes. (Figure 2) Color doppler imaging can detect the presence of blood flow within the walls of the affected bowel, demonstrating viable tissue.[9] (Figure 3) Absence of flow indicates infarcted bowel.[10] Occasionally, a lead mass is present within the dilated bowel. A small amount of free fluid can be a normal finding provided it does not contain debris, and visualization of fluid should not preclude non-surgical reduction.[11]

Although we still use radioopaque contrast or air with fluoroscopic guidance to hydrostatically reduce intussusceptions at our institution, water with ultrasound guidance has also successfully been utilized.[12] In any method of reduction, the head of the intussusceptum must be seen to pass through the ileocecal valve. The contrast material (or air) should then be seen to freely reflux into the distal small bowel.

Pneumoperitoneum is an absolute contraindication to attempted reduction and should be easily detected on the left lateral decubitus radiograph. The presence of peritonitis is another contraindication; for this reason, the surgical service should be consulted prior to any reduction.

ACUTE APPENDICITIS

Acute appendicitis is one of the most common causes of abdominal pain in children, with a high incidence between the ages of 4 and 15 years. The classic presentation usually begins with periumbilical and epigastric pain, followed by anorexia and later vomiting. In time, the pain shifts to the right lower quadrant. There can also be pain with walking or standing, indicating a retrocecal appendix.[13]

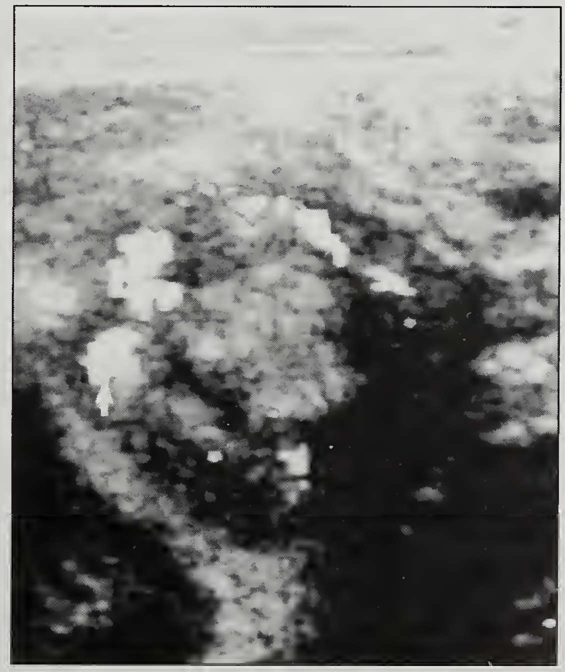

Figure 3: Color Doppler signal (arrow) demonstrates good blood flow and presumably viable intestine.

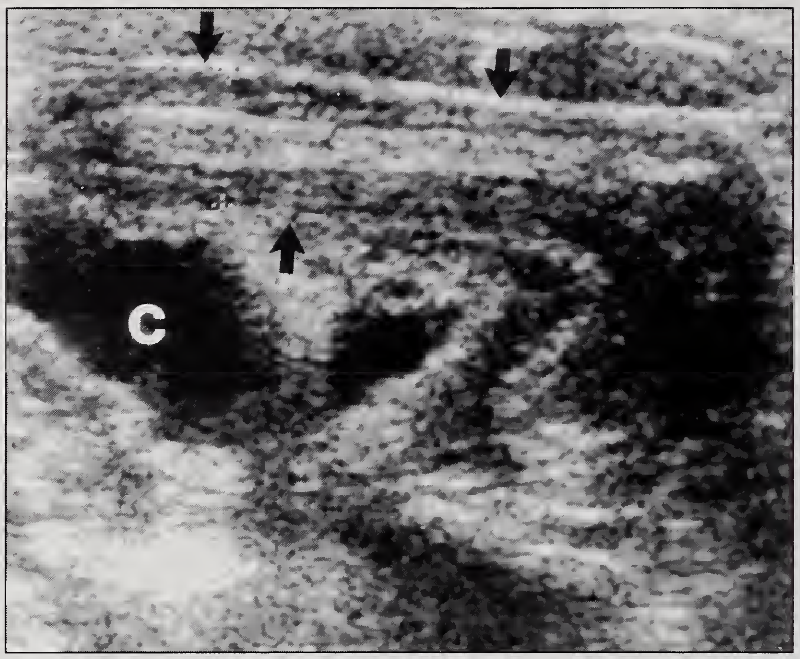

Figure 4: Sonogram of the right lower quadrant. Arrows delineate a surgically-proven acute appendix. This structure was non-compressible and measured 6.1 mm. The patient was also quite tender to graded compression over this area. The adjacent fluid-filled structure is the cecum (c).

It is important to establish the diagnosis promptly, as delay can lead to rupture, abscess formation and peritonitis. Appendiceal rupture can result in longer hospitalizations and for girls, possible fertility problems.[14] The diagnosis can be difficult because the presentation can be variable, and laboratory tests are often equivocal. Children without classic exam findings often proceed to imaging.

Non-contrast radiographs (KUB) can show a gasless abdomen secondary to vomiting and diarrhea. Air-fluid levels, especially in the right lower quadrant, and calcified appendicoliths can also be seen. This latter finding, while specific, is present in only approximately 15% of cases. One can also sometimes see a lumbar scoliosis concave to the right, or loss of the right psoas margin.[15] When findings are equivocal, US is the principal modality utilized in children. US can also sometimes discover an alternative etiology of the symptoms, particularly in girls where the pain has a gynecological origin. CT can be a helpful adjunct to evaluate complicated cases, but it is not the initial study of choice. This prioritization of US over CT is not necessarily true in the adult where the larger size of the patients and the increased amount of intraabdominal fat have made CT the first-line modality.

A contrast enema has been the traditional method to diagnose intussusception, but US has begun to supplant it as a screening modality.

The method of US examination was first described by Puylaert in 1986.[16] Normal bowel loops, including the appendix, compress with pressure. An inflamed appendix will not compress. During an exam, pressure is applied gradually, a technique known as graded compression. It is important to visualize the viscera down to the psoas muscle and iliac vessels. An abnormal appendix will appear as a tubular, non-compressible structure at least 6 mm in diameter. (Figure 4) Other possible findings include an appendicolith, which will appear as an echogenic focus, or a focal fluid collection indicating a periappendiceal abscess. The appendix must be visualized to its tip, as the inflammation can be confined to this area.[17] It is helpful to have the patient point to the spot of maximum tenderness to expedite the search for an inflamed appendix and reduce the time of the exam.[18] The absence of a normal appendix does not exclude the diagnosis, as only a minority of normal appendices are seen on exam.

Other gastrointestinal tract abnormalities and renal problems can mimic appendicitis.[19] Mesenteric lymphadenitis was the most frequent diagnosis of children who went to surgery for suspected acute appendicitis and were subsequently found to have a normal appendix.[20] Graded compression US can reveal the presence of enlarged mesenteric lymph nodes, which are considered abnormal when the maximum anteroposterior diameter is at least 4mm.[14] The presence of abnormal nodes alone should not, however, deter the sonographer from searching for addition findings. The finding of lymphadenitis is a non-specific marker of inflammatory, infectious , or neoplastic disease in the abdomen or pelvis.

MIDGUT VOLVULUS AND OTHER CAUSES OF BILIOUS VOMITING

The presence of bilious vomiting in a newborn is an absolute emergency, as it may indicate midgut volvulus in a malrotated infant. The twisting of the narrow mesenteric pedicle which contains the superior mesenteric artery can rapidly progress to infarction of the entire small bowel and the proximal portion of the colon. Plain films may reveal a gasless abdomen or evidence of a small bowel obstruction. The classic "double bubble" of a dilated, air-filled stomach and duodenal bulb is only infrequently seen. Suggestive plain film findings in the proper clinical setting are diagnostic, and surgery should not be delayed for further imaging.[21]

US has been suggested as an alternative to the upper GI in the cases where the diagnosis remains in doubt. On the upper GI, the position of the Ligament of Treitz, i.e. the duodenojejunal junction, should be at least as left as the left vertebral pedicle on a non-obliqued anteroposterior radiograph, and as least as superior as the duodenal bulb. A normally-placed ligament indicates normal mesenteric fixation. Because the embryological error leading to most cases of volvulus is failure of the last 180° of rotation of the midgut, the superior mesenteric artery, instead of being in its normal location to the left of the superior mesenteric vein, is located to the right. This relationship is usually easy to determine by US. The problem is that as many as one third of surgically proven malrotations had normally related mesenteric vessels on US. There are also cases of patients with normally fixated bowel, but inverted mesenteric vessels.[22] Therefore, US cannot be used as the primary modality to screen for malrotation, although the diagnosis can be suggested on an exam ordered for other reasons. Upper GI remains the gold standard.

SUMMARY

As equipment and techniques in ultrasonography improve, its role in the evaluation of infants and children has increased. Besides the traditional uses in the solid viscera and biliary tract, US can be a primary modality in the workup of pediatric gastrointestinal disease.

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Imaging the Patient with Acute Abdominal Pain: Current Concepts

William W. Mayo-Smith, M.D.

Acute abdominal pain is a common patient complaint. With the acknowledged lack of specificity of the physical exam, radiologic studies have played an increasingly important role in the diagnosis of abdominal pathology. Radiologic examinations available to the referring physician include the non-contrast radiograph, computed tomography (CT), ultrasound, nuclear medicine, and in the appropriate clinical setting, angiography and magnetic resonance imaging. There has been a revolution in the radiologic evaluation of patients with abdominal pain in the past five years with improved rates of accuracy and detection of disease. The purpose of this article is to describe new imaging techniques for the evaluation of patients with abdominal pain. In addition, this discussion will serve as a guide to ordering the most appropriate radiologic exam for a patient presenting with abdominal pain. *It is critical that the referring physician understand that the selection of CT or ultrasonographic examinations and the manner in which they are performed depend on the suspected clinical diagnosis.* Consultation with the radiologist is essential to perform the best radiologic exam for optimal patient care.

The primary reason for recent advances in imaging accuracy in the evaluation of abdominal pain is improved ultrasound and CT scan technology. The resolution of ultrasound has improved dramatically with advances in transducer technology, such as Doppler, power Doppler and most recently tissue harmonics. These advances have provided better imaging resolution and allowed visualization and characterization of tissue vasculature. Progress in CT has been contingent upon the development of helical scanning technology. Helical CT acquires image data continuously while the patient is advanced through the scanner; thus, the image data is a volume, not a group of individual slices. The advantage of this technology is two-fold. First, the data is acquired more rapidly, the entire abdomen and pelvis can be imaged during one breathhold. Second, the rapid data acquisition prevents spatial misregistration or movement between slices. Thus, pathology is not "missed" between images. In addition, helical CT has permitted the development of CT angiography where blood vessels can be imaged directly without having to perform an angiogram.

Traditionally, supine and erect non-contrast radiographs of the abdomen historically termed a KUB (an abbreviation for kidneys, ureter and bladder) have been the most commonly performed radiologic examination for evaluation of abdominal pain. This technique is reliable in detecting free intraperitoneal air following a perforated viscus and can occasionally detect urinary calculi and bowel obstruction. However, renal calculi are often not visualized or can be confused with phleboliths. Abscesses are not reliably detected and bowel obstruction is only visualized if the dilated small bowel loops are air filled. Recent radiologic literature has demonstrated improved accuracy in detecting abdominal pathology using CT and ultrasound compared with a non-contrast radiograph as outlined for the following disease states.

Abbreviations Used:

BE	barium enema
CT	computed tomography
IVU	intravenous urogram
KUB	kidneys, ureter and bladder

Appendicitis

Appendicitis is a difficult clinical diagnosis evidenced by the high negative laparotomy rate reported in the medical and surgical literature. There has been improved accuracy in the diagnosis of appendicitis using both ultrasound and more recently CT. The normal appendix can be visualized with a focused CT examination in up to 100% of patients. In a recent article in the *New England Journal of Medicine*, Rao et al reported 98% overall accuracy of CT in establishing the diagnosis of appendicitis. When considering the cost savings from an immediate laparotomy or discharge vs. admission and observation, Rao et al

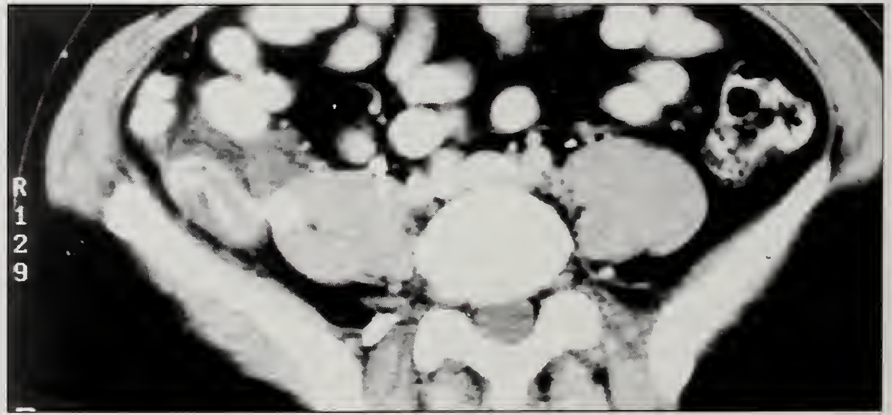

Figure 1. A 38-year-old man with right lower quadrant pain. CT with oral and intravenous contrast demonstrates a dilated, inflamed appendix (arrow). The CT scan is diagnostic for appendicitis and the finding was confirmed at surgery.

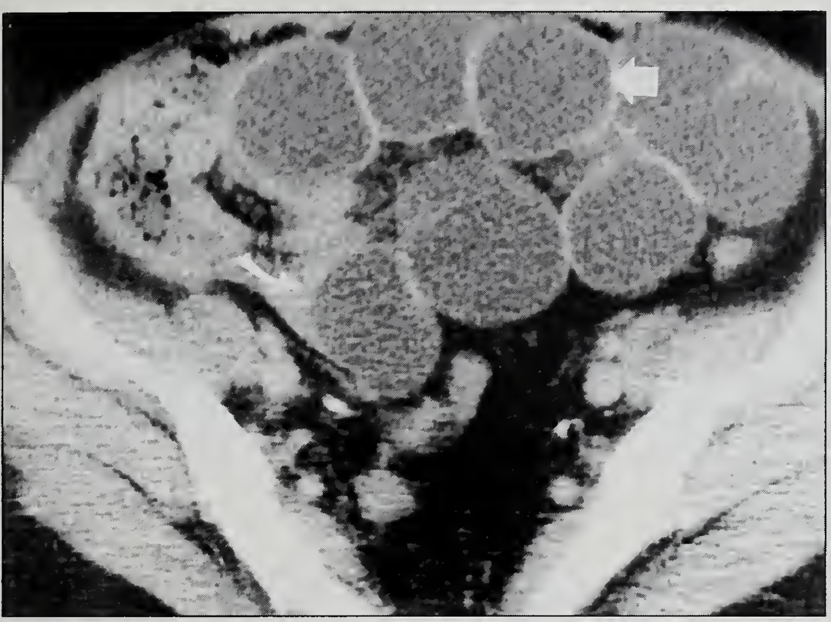

Figure 2. A 55-year-old woman with a history of ovarian carcinoma. Non-contrast abdominal radiographs were equivocal for small bowel obstruction. CT demonstrated fluid-filled dilated loops of small bowel (arrows) with peritoneal implants from recurrent ovarian tumor causing the obstruction (curved arrow).

calculated a cost savings of $447 per patient when performing CT for the suspected diagnosis of appendicitis.[1] Many articles supporting the utility of CT in diagnosing patients with suspected appendicitis have appeared in the medical literature.[2-4] The most accurate results for diagnosis of appendicitis have been performed using contrast administered per rectum, although this route of administration is controversial. Depending on the clinical indication, intravenous contrast agents can be administered at the discretion of the radiologist. A CT diagnosis of appendicitis can be made when an inflamed, dilated appendix is visualized. (Figure 1)

Bowel Obstruction

Small bowel obstruction in a patient with nausea and vomiting and appropriate history can often be made on the basis of the physical exam. Non-contrast radiographs are useful to determine whether a small bowel obstruction exists but their negative predictive value is less reliable. Small bowel obstruction on radiographs is diagnosed when there are numerous dilated air-filled loops of small bowel with a relative paucity of bowel gas within the colon. The shortfall of these non-contrast radiographs occurs when the small bowel is fluid-filled and not visualized on radiographs.

In 1991, Megabow et al, published a landmark article on the utility of CT in diagnosing the presence and etiology of small bowel obstruction.[5] In this study, the CT examinations of 84 patients with a clinical history questioning bowel obstruction were compared with 83 controls. CT had a sensitivity of 94%, a specificity of 96% and an accuracy of 95%. The etiologies of small bowel obstruction in this cohort were adhesions (37), metastases (6), primary bowel tumor (7), Crohn's disease (4), hernia (3), hematoma (2), diverticulitis and other.[5] Numerous subsequent publications have confirmed these results.[6-8] CT is useful for detecting not only the presence of obstruction but also its underlying cause. Inflammatory bowel disease, primary neoplasm, hernia and adhesions may all have different surgical approaches. Thus, the ability of CT to define not only the presence but also the etiology of small bowel obstruction impacts significantly on patient management. In general, oral contrast is not necessary when performing CT in these patients as the fluid-filled loops of obstructed bowel are adequately visualized in the axial plane. (Figure 2)

It is critical that the referring physician understand that the selection of CT or ultrasonographic examinations and the manner in which they are performed depends on the suspected clinical diagnosis.

Diverticulitis and Inflammatory Bowel Disease

Diverticulitis increases in frequency with age and is estimated to occur in 80% of the population over 85 years. Approximately 20% of patients with diverticulosis will develop diverticulitis at some point in their life.[9] The majority of patients with diverticulitis present with left lower quadrant pain but they may have right-sided pain from right colonic diverticulitis or secondary to a redundant sigmoid colon projecting over the cecum. These latter patients are difficult to separate clinically from patients with appendicitis yet the treatment of these two groups of patients varies significantly. While barium enema (BE) was originally used to establish a diagnosis of diverticulitis, CT has replaced BE as it is more sensitive and specific with fewer complications.[10,11]

Since diverticulitis (inflammation of the diverticula with perforation) is primarily an extra-luminal disease, CT is useful to not only establish a diagnosis, but also to stage the disease. Stage I represents microperforation with inflammation around the diverticula and stage IV represents frank perforation with fecal peritonitis. After the diagnosis of diverticulitis has been established by CT, imaging-guided catheter placement in patients with a focal intra-abdominal abscess can be performed by the radiologist.[12] In combination with systemic antibiotics, percutaneous drainage can control sepsis and allow a one-stage surgical

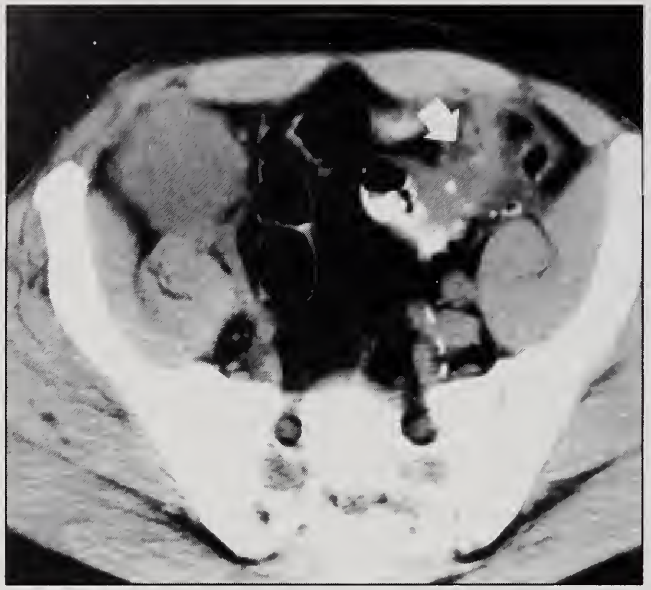

Figure 3. A 49-year-old male with left lower quadrant pain and fever. The CT with oral contrast demonstrated thickening of the sigmoid colon with stranding of the peri-colonic fat (arrow). The CT findings are consistent with diverticulitis.

procedure to resect the involved segment, thereby avoiding a second operation and temporary colostomy. (Figure 3) A false negative rate of 2-20% has been reported with CT and sigmoid carcinoma can mimic findings of diverticulitis on CT.[13] Patients who do not improve symptomatically following the administration of systemic antibiotics should undergo a barium enema or sigmoidoscopy to exclude the possibility of underlying carcinoma.

Computed tomography is the study of choice for evaluating patients with known or suspected Crohn's disease presenting with severe abdominal pain.[14] CT is useful in determining the site of inflammation, the presence of an extra-luminal abscess and to exclude other sites of disease. Small bowel barium studies and barium enema are more sensitive for locating the presence and site of enteric fistulas.[15]

GYNECOLOGIC PATHOLOGY

If a gynecologic source is the presumed etiology of abdominal pain, ultrasound is the imaging modality of first choice. If ectopic pregnancy is a clinical consideration, a beta HCG should be performed to determine pregnancy followed by a transvaginal ultrasound. The most useful ultrasound finding to exclude an ectopic pregnancy is the presence of an intrauterine gestation. Ultrasound can reliably detect ovarian size, differentiate solid from cystic masses, and detect fluid in the cul-de-sac. Ovarian cysts are reliably imaged and a ruptured follicle at the time of ovulation can be inferred if the pain presents at the correct time in the menstrual cycle and there is fluid in the cul-de-sac on transvaginal ultrasound. Ovarian torsion is a difficult diagnosis to establish both clinically and by imaging studies. The most reliable feature is an enlarged ovary on pelvic ultrasound. Doppler ultrasound has not been as reliable at predicting ovarian torsion as originally hoped. This is probably due to the variable amounts of perfusion to the torsed ovary and the intermittent nature of torsion.

Ovarian carcinoma is a difficult diagnosis to establish clinically, has no reliable screening test, and most patients present with advanced disease. The patients often present with nonspecific abdominal pain and bloating. On abdominal CT, a large pelvic mass can be seen with ascites and omental cake which suggests the diagnosis.[16] Ultrasound guided biopsy or paracentesis can be performed to confirm a malignant etiology in these patients. In general, cervical neoplasms are diagnosed by physical exam. Endometrial carcinoma is suspected in a post-menopausal woman who has new onset of menstrual bleeding. Diseases of the fallopian tubes such as tubo-ovarian abscess are diagnosed by physical exam, laboratory findings and the presence of enlarged tubular adnexal masses on pelvic ultrasound.

KIDNEY STONES

Patients with a urinary tract stone who present with the classic symptoms of acute flank pain and hematuria rarely present a diagnostic dilemma. However, many patients present with less obvious clinical signs. In addition, the treatment options for patients presenting with nephrolithiasis has expanded significantly in recent years and includes extracorporeal shock-wave lithotripsy, endoscopic basket retrieval, endoscopic ultrasonic lithotripsy, endoscopic laser lithotripsy, percutaneous lithotomy, stent placement or expectant management. The myriad of treatment options open to the urologist depends on clinical presentation, coexisting medical history and the composition, size and location of the renal

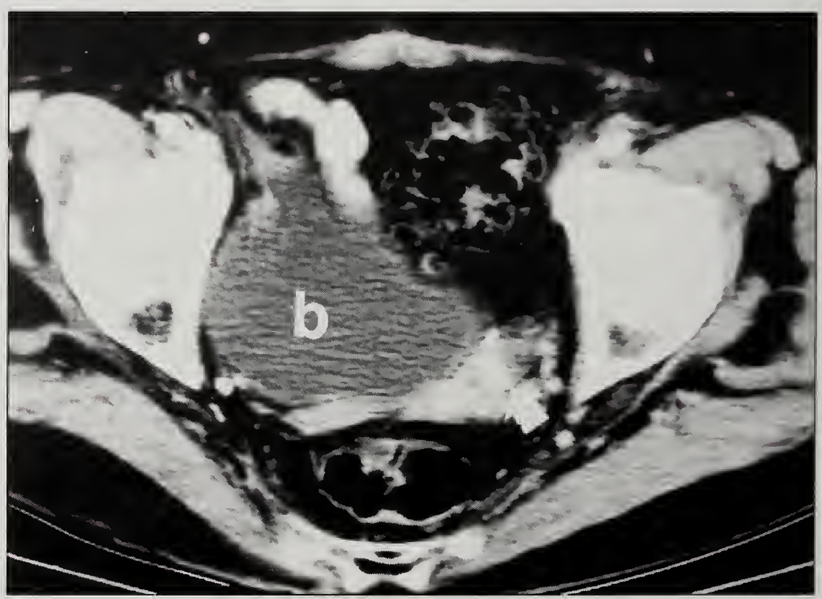

Figure 4. An 80-year-old woman with left flank pain. A CT with oral but not intravenous contrast demonstrates a radiopaque calculus in the distal left ureter near the ureterovesicular junction (arrow). (b=bladder) More cranial images in this patient (not shown) demonstrated hydronephrosis and hydroureter.

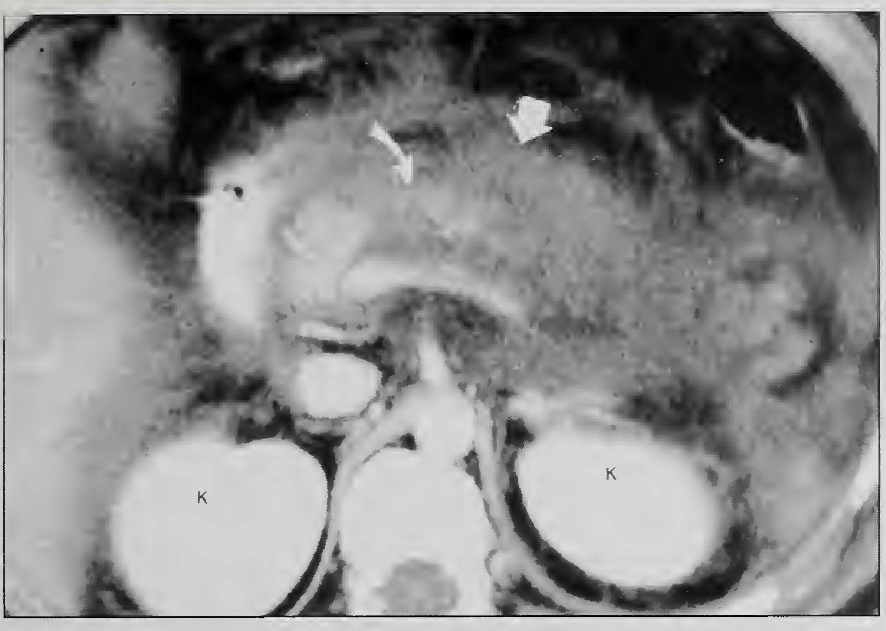

Figure 5. A 24-year-old man with a history of alcohol abuse and four days of abdominal pain. CT scan with oral and intravenous contrast demonstrates enlargement of the pancreas with inflammation of the peri-pancreatic fat (arrow) diagnostic for acute pancreatitis. In addition, there is pancreatic necrosis present (curved arrow) which is a poor prognostic factor for patients with acute pancreatitis. This patient required subsequent surgical debridement of the pancreas. (K=kidney)

calculi.

Traditionally, an intravenous urogram (IVU) has been performed to evaluate for suspected renal calculi and it remains useful for certain indications. The advantage of the intravenous urogram is that it demonstrates renal function, the level of obstruction, and can occasionally be therapeutic as the high osmolarity of the intravenous contrast induces a diuresis which propels the ureteral calculi into the bladder. The disadvantages of intravenous urogram are its expense and the inherent risks and costs of intravenous contrast material. In 1995, Smith et al published a manuscript comparing the accuracy of non-contrast helical CT with intravenous urograms in 20 patients with acute flank pain where ureteral obstruction was suspected.[17] They found that noncontrast CT was more effective in determining the presence of a calculus (11/12 patients) than intravenous urogram (6/12 patients) and was equally effective at determining the presence or absence of obstruction. (Figure 4) Numerous manuscripts subsequent to this have confirmed the accuracy of nonenhanced helical CT and its role in management of renal calculi.[18-20] In an interesting article analyzing the role of helical CT in planning treatment of 100 patients with proven ureteral calculi, Fielding et al found that calculi larger than 5 mm., located in the proximal 2/3 of the ureter and seen on two consecutive CT images were more likely to require endoscopic removal, lithotripsy, or both.[21] CT scan determines obstruction by detecting dilatation of the proximal ureter and collecting system. CT is also useful in detecting a urinoma around the kidney. The advantage of CT in a patient presenting with flank pain is that the exam is rapidly performed, does not involve the use of intravenous or oral contrast and is useful to diagnose other intra-abdominal entities that may mimic urolithiasis. The disadvantage is a slightly higher radiation dose to the patient and increased cost.

PANCREATIC AND BILIARY DISEASE

Ultrasound remains the study of choice for patients with presumed cholecystitis. Ultrasound is easy to perform and does not require ionizing radiation. Ultrasound is also useful to assess for intrahepatic biliary dilatation in patients with presumed cholangitis. CT is the imaging technique of choice for presumed pancreatitis. (Figure 5) CT can reliably image the pancreas, detect peripancreatic inflammation, determine if there is pancreatic necrosis present or image the sequelae of chronic pancreatitis (dilated pancreatic duct, intrapancreatic calculi, pseudo-cyst and ascites).[22] Ultrasound is less reliable for detecting pancreatitis as perfusion is difficult to assess, the mesentery is not reliably imaged and the exam may be limited by overlying bowel gas or obesity.

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Other Diseases

For patients with a known abdominal aortic aneurysm in whom a leak is suspected, CT is the study of choice. CT can image the retroperitoneum particularly when looking for a retroperitoneal bleed without the administration of oral or intravenous contrast. The examination can be performed in minutes without any advance preparation.

Summary

The development of improved imaging technology, specifically development of helical CT and improvement in ultrasound, has revolutionized the work-up of patients with abdominal pain.[23] It is important to remember that the type of examination performed depends on the suspected clinical diagnosis. In particular, the CT exam prescription by the radiologist varies significantly depending on the type of pathology suspected. The route of the administration of gastrointestinal contrast (oral or per rectum), the use of intravenous contrast, slice collimation, the field of view and other parameters can significantly improve the accuracy of the study. Thus, accurate communication with the examining physician is critical. When used properly, radiologic examinations can significantly impact on patient care and management.

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Innovations in Neuroimaging and Neurointerventional Radiology

Glenn A. Tung, MD, Lawrence M. Davis, MD, Jeffrey M. Rogg, MD, Richard A. Haas, MD, Richard L. Gold, MD

As we near the end of the "Decade of the Brain," it is fitting to review a few of the salient innovations in MR imaging and interventional radiology that are being used to diagnose and treat neurologic disease. Herein, we will discuss MR hydrography, diffusion- and perfusion-weighted imaging, magnetic resonance spectroscopy, and the neurointerventional treatment of cerebral aneurysms with detachable coils.

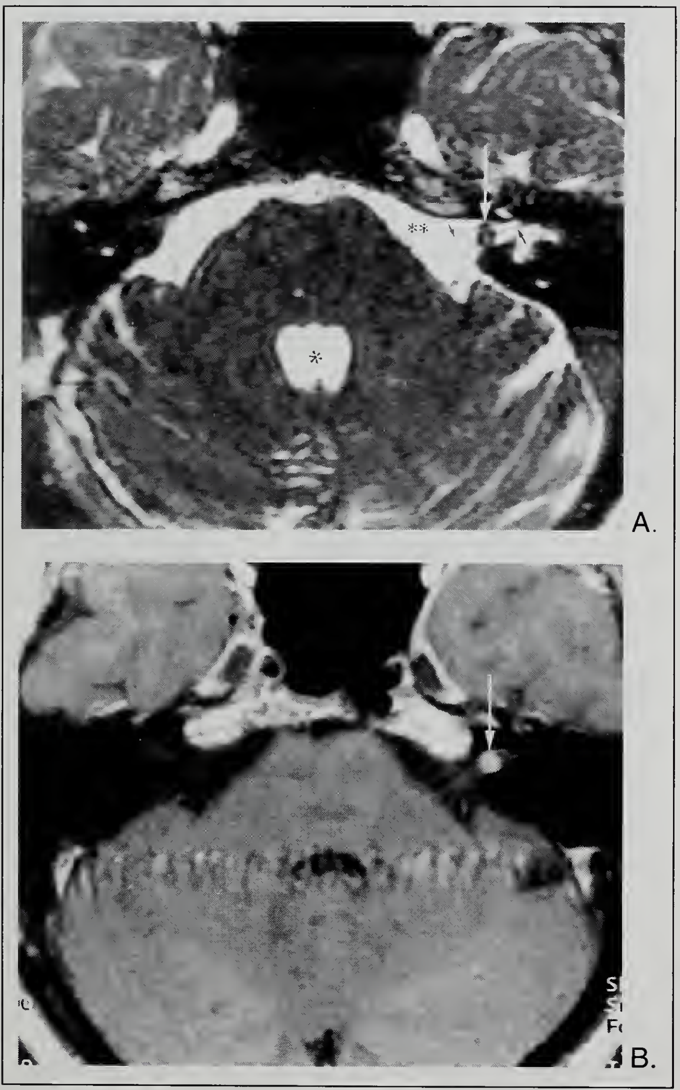

Figure 1. Intracanalicular vestibular schwannoma is demonstrated on both thin-section MR cisternography and standard enhanced-MR imaging. MR cisternogram (A) at the level of the cerebellopontine angle (CPA) cistern shows a 3 mm hypointense mass in the left internal auditory canal (white arrow). Cerebrospinal fluid in the fourth ventricle (*) and CPA cistern (**) is hyperintense compared to the cerebellum and brainstem. Note that the left facial and superior vestibular cranial nerves (small black arrows) in the internal auditory canal can be resolved by this technique since the thickness of this slice is 0.7mm. Contrast-enhanced, T1-weighted image (B) of the same patient shows the typical appearance of an intracanalicular vestibular schwannoma.

Abbreviations Used:

CT	computed tomography
GDC	Guglielmi detachable coil
IAC	internal auditory canal
MRH	magnetic resonance hydrography
MRS	magnetic resonance spectroscopy
NAA	N-acetyl aspartate
SAH	subarachnoid hemorrhage

MAGNETIC RESONANCE HYDROGRAPHY

Magnetic resonance hydrography (MRH) refers to a group of MR imaging techniques that have been used to image ducts, hollow viscera, or other body spaces that predominately contain "simple fluids." To date, this technique has been used at the Rhode Island and Miriam Hospitals to evaluate the biliary and pancreatic ducts, and the renal collecting system. In the head and neck, MR hydrography has been applied to imaging the intracranial cisterns and internal auditory canals (MR cisternography), membranous labyrinth (MR labyrinthography), and salivary gland ducts (MR sialography).[1-7]

The most common application of MRH in the head and neck has been for the detection of acoustic neuroma or vestibular schwannoma. Since the normal internal auditory canal (IAC) is less than 1cm in diameter and contains four cranial nerves (facial, cochlear, superior vesticular, and inferior vestibular) that are each approximately 1 mm in diameter, MR cisternography of the IAC requires high-resolution images. On these images, cranial nerves and masses appear as dark filling defects within bright, white CSF.[2] Since the late eighties, MR imaging with gadolinium contrast has been the gold standard for the detection of vestibular schwannomas. Recent work has shown that high resolution MR cisternography can detect masses as small as 2mm in the IAC with a sensitivity comparable to contrast-enhanced MR imaging.[3] (Figure 1) With MR cisternography, one could screen patients with suspect acoustic neuroma more quickly, less expensively, and less invasively than traditional enhanced-MR imaging. However, additional study is needed before this technique replaces the gold standard.

MR labyrinthography produces high-resolution two- and three-dimensional images of the membranous labyrinth.[4] Some centers use this technique to evaluate hearing loss related to the labyrinth, although it has not yet replaced high-resolution computed tomography (CT). The fluid (perilymph and endolymph) of the membranous labyrinth appears very bright within the black background of the bony labyrinth. Two common congenital causes of hearing loss

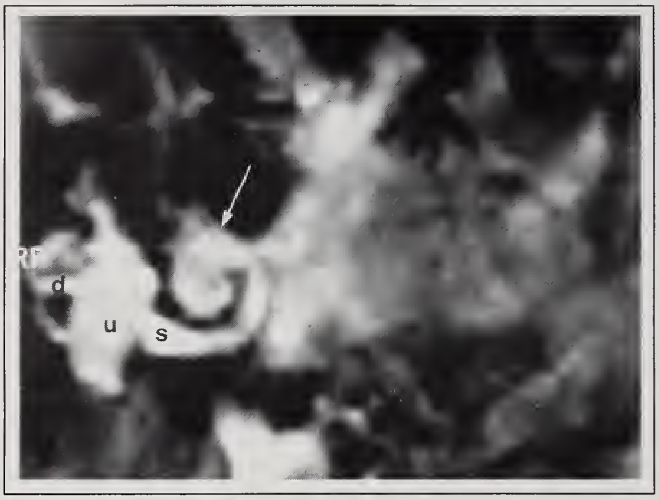

Figure 2. MR labyrinthogram demonstrates Mondini malformation of the right inner ear. Since the membranous labyrinth contains endolymph, a "simple" fluid, it appears hyperintense on the heavily T2-weighted images used for MR hydrography. The normal cochlea is a coiled structure consisting of two and one-half turns but in this case, the cochlear duct has only one turn (arrow). In addition to the cochlea, the membranous labyrinth consists of the saccule (s), utricle (u), semicircular ducts (d), and the endolymphatic duct.

are the dilated vestibular aqueduct syndrome and Mondini malformation. In dilated vestibular aqueduct syndrome, MR labyrinthography can demonstrate both the enlarged aqueduct and the dilated endolymphatic sac, whereas CT only shows the large vestibular aqueduct. MR labyrinthography also produces a three-dimensional representation and routinely shows the normal two and one half turns of the cochlea.[5,6] The Mondini malformation is a congenital inner ear anomaly in which there are too few turns of the cochlea and has been demonstrated by MR labyrinthography. (Figure 2)

The most recent application of MRH in the head and neck is MR sialography. This technique produces images that are similar to iodinated-contrast sialography in which the fluid-filled salivary ducts are bright white against darker background tissues. However, MR sialography is noninvasive and requires neither ionizing radiation nor iodinated contrast. Recent reports have shown the feasibility of this technique for evaluating the parotid and submandibular ducts and MR sialography has been used to evaluate parotid tumors, duct calculi and strictures, and Sjögren syndrome.[7]

DIFFUSION- AND PERFUSION-WEIGHTED MR IMAGING

The treatment of ischemic stroke is changing. Thrombolysis and the development of neuroprotective agents have given rise to the national "brain attack" initiative which intends to raise the consciousness of physicians, paramedical personnel, and patients to the urgency of an early diagnosis and treatment of ischemic stroke. For the accurate and timely diagnosis of acute stroke, diffusion- and perfusion-weighted MR imaging have become essential diagnostic tools.

Molecular diffusion is the random, translational movement of small molecules in tissue that is caused by thermal processes, like Brownian motion. Experimental data suggests that injured, ischemic brain shows marked changes in water diffusion because extracellular water is shifted to the more diffusion-limited intracellular space when cellular ion

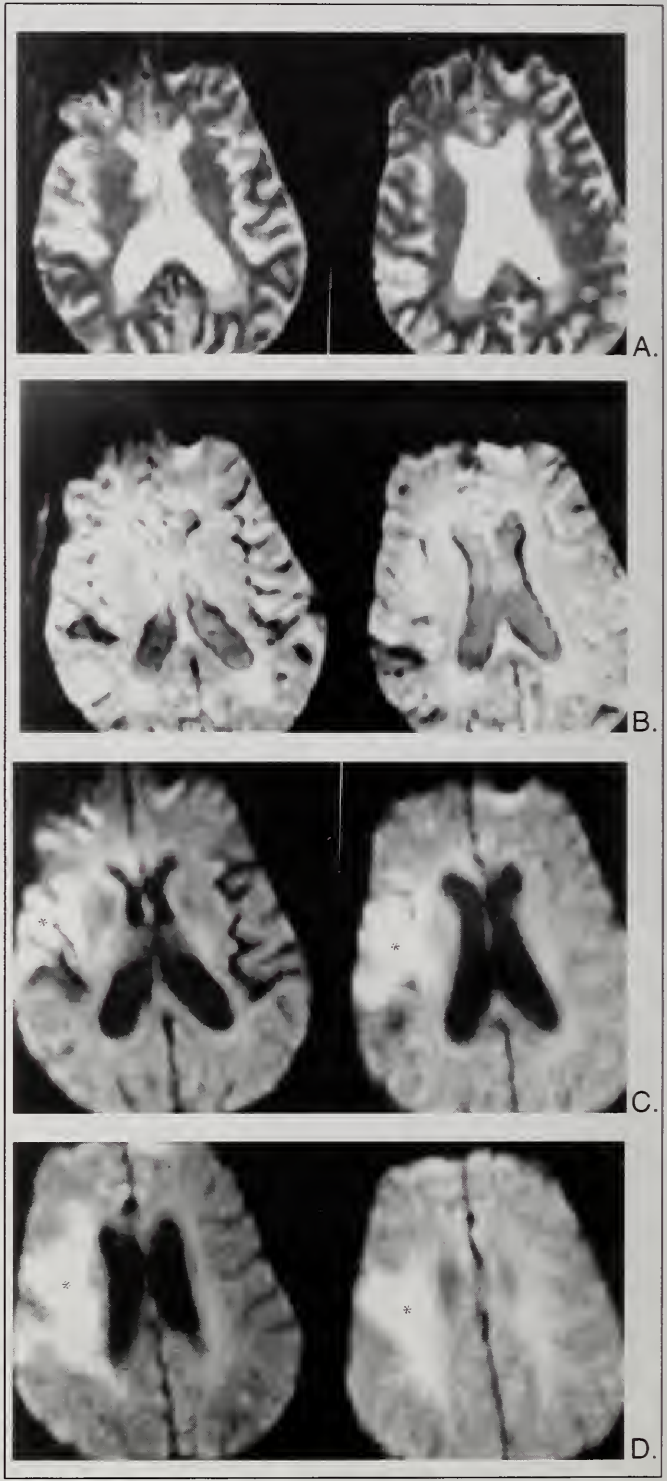

Figure 3. Diffusion-weighted MR imaging is more sensitive than conventional MR imaging (fluid-attenuated inversion recovery and T2-weighted images) for the early detection of ischemic stroke. An elderly woman presented approximately four hours after the onset of nonspecific neurologic symptoms. Echo-planar turbo-T2-weighted (A) and fluid-attenuated inversion recovery (B) images show cerebral atrophy and periventricular white matter changes due to small vessel disease. Diffusion-weighted images (C, D) obtained at the same time demonstrate extensive increased signal intensity in the cortex and subcortical white matter (marked with *) in the right middle cerebral artery vascular distribution consistent with acute brain ischemia.

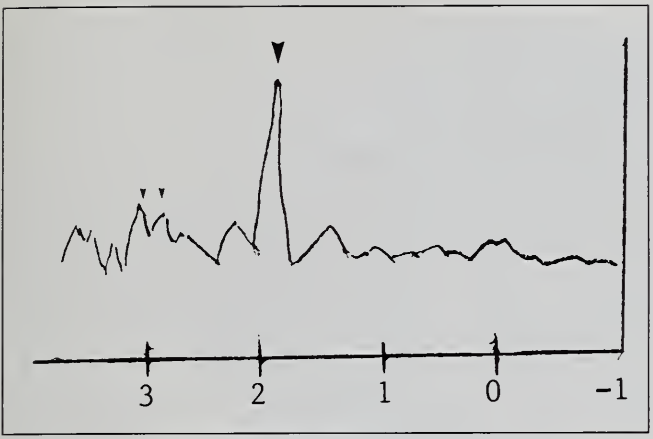

Figure 4. Normal proton MR spectrum of the brain. The large peak at 2.0ppm is assigned to N-acetyl aspartate (NAA-large arrow head). Smaller peaks at 3.2 ppm and 3.0 ppm are assigned to choline and creatine respectively (small arrow heads).

pumps fail.[8] With special diffusion-sensitive pulse sequences, diffusion-weighted MR imaging shows regions of brain with fast water diffusion as areas of low signal intensity and slow diffusion areas as regions of relatively higher intensity. In animal models of stroke, decreased water diffusion was demonstrated within minutes after an ischemic insult.[10] In human studies, diffusion-weighted imaging has been shown to be more sensitive than conventional MR imaging for demonstrating ischemic brain injury within the first six hours after the onset of stroke symptoms.[11] (Figure 3)

Perfusion is measured by the volume of blood delivered to a mass of tissue per unit of time. A decade ago, it was proposed that MR imaging could be used to measure perfusion by mapping the distribution and concentration of a blood-borne contrast agent as it passes through a tissue of interest. One would expect that the signal intensity of normally perfused brain tissue to change as the intravascular contrast agent passes through it. Investigations to date suggest that, in contrast to infarcted brain, tissue in which ischemia is fundamentally reversible-the "penumbra"-shows reduced perfusion but normal tissue diffusion. Thus the combination of diffusion- and perfusion-weighted MR imaging may distinguish irreversibly injured ischemic tissue from potentially salvageable "tissue at risk"; this distinction has critical implications for the development and study of therapeutic stroke interventions.[12]

Magnetic Resonance Spectroscopy

Magnetic resonance spectroscopy (MRS) is a technique that extends the power of MR imaging to an analysis of the chemical constituents of normal and abnormal tissue. MRS relies on the spin of nuclei when they are exposed to an external magnetic field. The spin of particular nuclei will have a distinctive phase and frequency that is determined by its chemical microenvironment. The MR spectrum is a biochemical fingerprint of a tissue based on its characteristic spins. The location and size of various "peaks" in the spectrum indicate the type and amount, respectively, of a particular biometabolite in that tissue. (Figure 4) Proton spectroscopy is an FDA-approved, non-invasive technique that can be performed in approximately 15 minutes.

Clinical research has shown that MRS of the brain can be used to evaluate both normal brain development and a wide variety of abnormal conditions. There are several metabolites that are commonly assayed. N-acetyl aspartate (NAA), the largest resolvable peak, is found only in the brain and is a marker of neuronal density and viability. Decreased NAA is found in a wide variety of insults to the brain such as infection, infarction, and tumor.[13] Decreased NAA may also serve as a marker for seizure foci or for neurodegenerative processes such as Alzheimer's disease.[14,15] Choline, the second largest peak, is a precursor for a major structural phospholipid of the cell membrane, phosphatidylcholine. The size of the choline peak reflects cell membrane synthesis, membrane integrity, and myelin formation. Increased choline has been noted in neoplasms, where there is increased cellular density and high membrane turnover (Figure 5), and in conditions where myelin breakdown has occurred.[16]

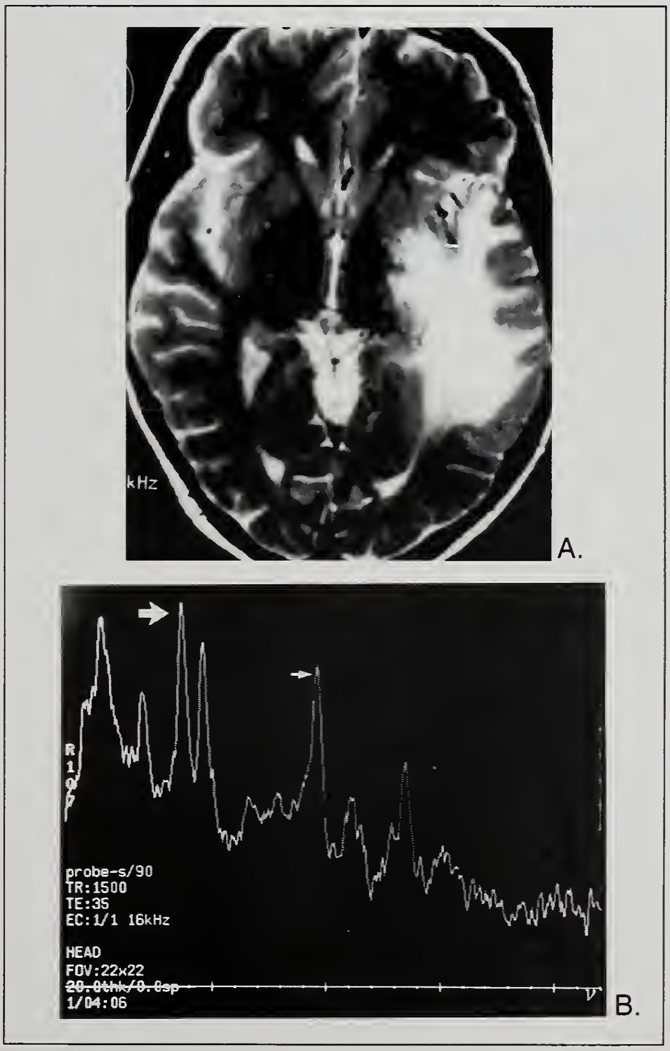

Figure 5. Proton MR spectroscopy of left insular glioma. Transaxial T2-weighted image image (A) demonstrates the sample volume for spectroscopic analysis in an area of abnormal signal in the left insula. A prominent choline peak (large arrow) in the MR spectrum (B) is attributed to increased membrane synthesis and cell density in glial neoplasms. The slightly decreased NAA peak (small arrow) is related to a reduction in normal neuronal elements in these tumors (Case courtesy of Lawrence M. Davis, MD).

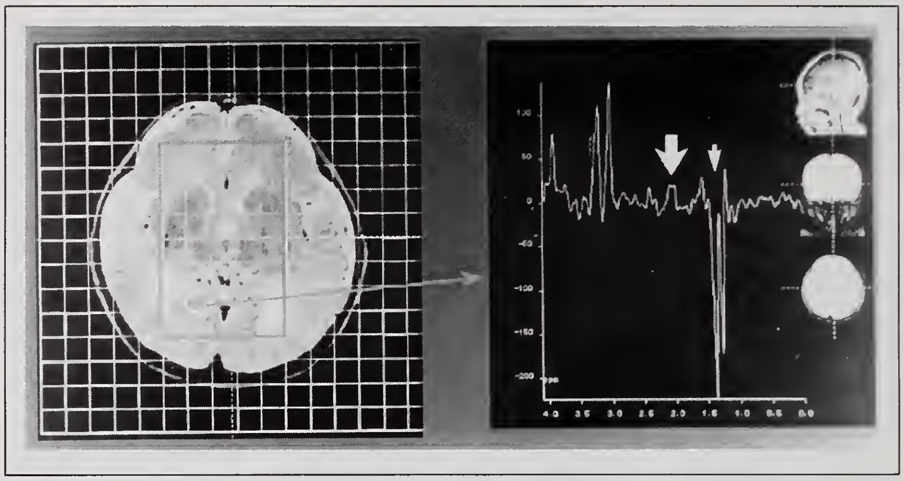

Figure 6. Proton MR spectroscopy of right occipital lobe ischemia. Transaxial image of the brain (left) demonstrates the sample volume in the right occipital lobe (small white square) for spectroscopic analysis. MR spectrum (right) demonstrates a decreased NAA peak (large arrow) resulting from loss of neurons due to infarction. Lactate doublet at 1.32ppm (small arrow) indicates that normal oxidative metabolism is no longer in effect. The lactate doublet is not present in the spectra of normal brain parenchyma (Case courtesy of Siemens Medical Systems).

Creatine, the third largest peak, plays a role in maintaining energy-dependant systems in the brain and decreases in creatine indirectly reflect cellular viability and will be decreased with brain ischemia.[16] Lactate has two peaks that are generally small or absent. The presence of lactate indicates energy metabolism by glycolysis that normally occurs in glial cells, macrophages, and lymphocytes. Thus, elevation of lactate levels may indicate activation of these cells and may be seen in inflammatory processes and demyelination.[16] The presence of lactate may also indicate a failure of oxidative metabolism and serve as a marker for ischemia (Figure 6). Other metabolites of clinical importance in MRS include myoinositol, glutamine, alanine and free lipids.[13]

In addition to the above applications, MRS has demonstrated early promise for specifying tumor histology, assessing the prognosis of multiple sclerosis, and evaluating neuropsychiatric disorders such as anorexia nervosa and schizophrenia.[17-20] Although MRS is not currently performed at the Rhode Island and Miriam Hospitals, we hope to make it available in the near future.

ENDOVASCULAR TREATMENT OF CEREBRAL ANEURYSMS

Subarachnoid hemorrhage (SAH) caused by aneurysm rupture is the third leading cause of stroke in the United States and about 130 cases are treated at the Rhode Island and Miriam Hospitals each year. The natural history of ruptured cerebral aneurysm is dire; up to 50% of patients die in the first two weeks. Half of these deaths are due to rebleeding and virtually all of these rebleeds are fatal. The risk of rebleeding in the first two weeks is about 20%, and the risk increases to 50% at six months for untreated ruptured aneurysms. Of those patients who survive the first six months, the annual rate of repeat SAH is approximately 3%.

> *Magnetic resonance spectroscopy (MRS) is a technique that extends the power of MR imaging to an analysis of the chemical constituents of normal and abnormal tissue.*

◆

Management of the patient with a ruptured cerebral aneurysm is directed at maintaining cerebral perfusion and preventing rebleeding. The mainstay of treatment is operative exploration and clipping of the ruptured aneurysm. Despite the 6% morbidity of early surgery, diminishing the frequency of second bleeds leads to better patient outcome (21). A surgically placed clip that completely obliterates the aneurysm prevents repeat hemorrhage from that aneurysm. In spite of near universal agreement in favor of early surgical intervention, some patients are not optimal candidates for clipping (22). A patient may be excluded from clipping if the location of the aneurysm limits surgical exposure (e.g., posterior fossa and cavernous sinus), if there is concomitant cerebral, or if there are comorbid conditions that increase the risk of craniotomy and general anesthesia.

Transcatheter embolization with detachable coils is a safe and effective alternative treatment for cerebral aneurysms when performed by an experienced neurointerventional radiologist and approximately twenty patients have been treated by this method in each of the last three years at the Rhode Island Hospital. The theory behind coil embolization is that, by filling the entire aneurysm lumen with a nest of metal coils, the coil mesh will ultimately incite thrombosis and over time a film of endothelium will form over the thrombosed neck of the aneurysm. Treatment is begun by advancing a microcatheter into the aneurysm. Coils are then pushed through the end of a microcatheter until the aneurysm is completely packed. The Guglielmi detachable coil (GDC) is attached to a stainless steel leader that permits more accurate coil deposition. Once the coil has been satisfactorily placed, a small electrical current is applied to the proximal end of the leader and the coil is electrolytically detached (Figure 7). Coils are available in various lengths and diameters as well as softness for optimal packing of the aneurysm lumen.

GDC embolization is particularly suited to aneurysms 4mm to 10mm in diameter with a small (less than or equal to 4mm) neck. It is less favorable in aneurysms with wide necks. The largest reported experience with this method to date has been from UCLA and includes a total of 327 patients with small, large, and giant aneurysms (23). Complete ablation of the aneurysm is reported in 92% of small aneurysms, and in 26% of giant

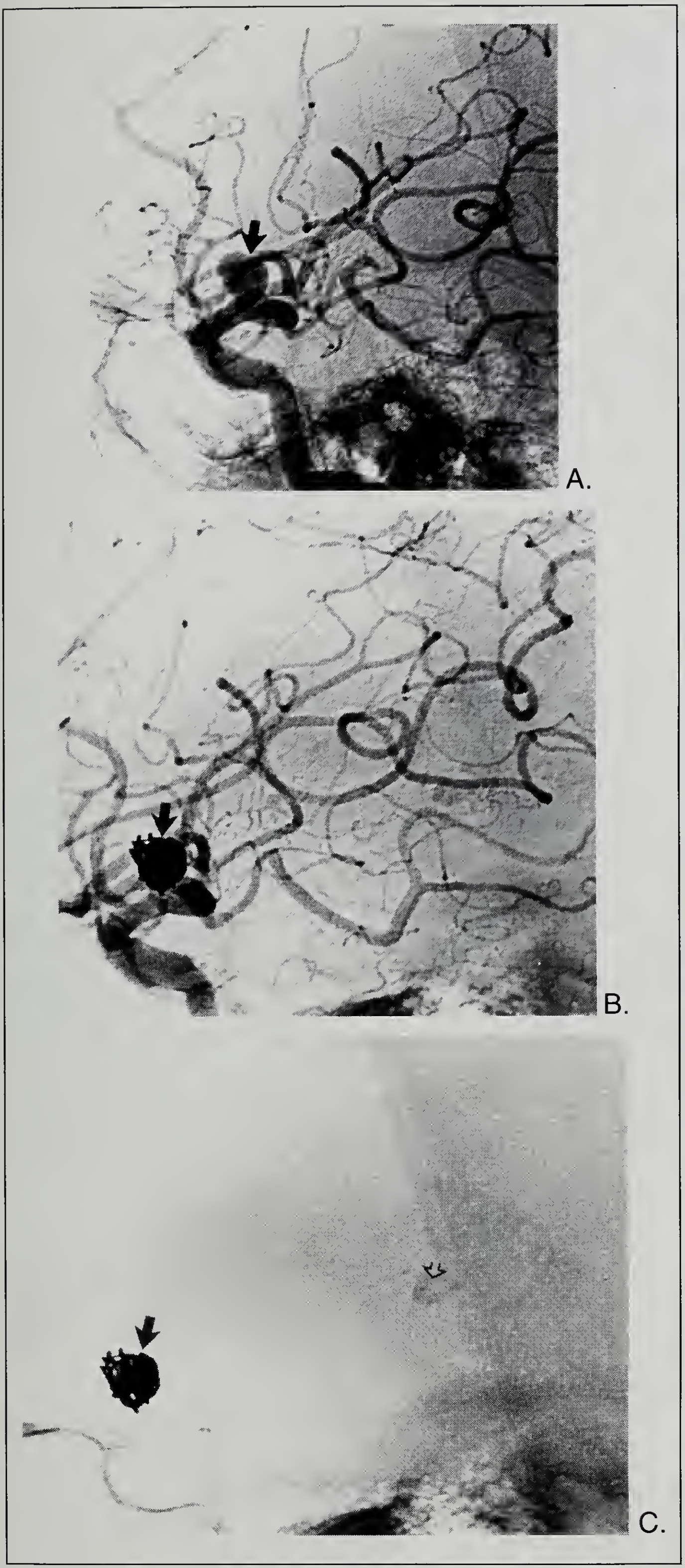

Figure 7. Endovascular treatment of a basilar tip aneurysm with Gugliemi detachable coils. A patient presented with acute subarachnoid hemorrhage. Subtraction cerebral arteriography (A) demonstrated a basilar tip aneurysm (arrow) that was not amenable to surgical clipping (b = basilar artery; * = subtracted dental fillings). Definitive endovascular treatment was accomplished by placing a microcatheter in the aneurysm lumen and progressively packing it with detachable coils. The post-procedure subtraction angiogram (B) demonstrates complete obliteration of the aneurysm and its neck

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aneurysms. Complications were reported in 7% of cases and included aneurysm perforation, distal embolization, and parent vessel occlusion. Among patients with good neurologic function (Hunt Hess Grades I and II), there was a 12% morbidity from this procedure and no deaths were reported. Among the more severely impaired patients (Hunt Hess Grades III, IV and V), the mortality rate was 10% and the morbidity rate was 8%. It is important to remember that morbidity related to the procedure may be difficult to separate from that associated with the natural history of SAH due to aneurysm rupture. In summary, endovascular treatment with detachable coils appears to be a viable alternative in patients who are not optimal surgical candidates.

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Radiofrequency Ablation: An Outpatient Percutaneous Treatment

Damian E. Dupuy, MD

Improvement in computed tomography (CT) and magnetic resonance imaging (MRI) scanners and ultrasound techniques have produced greater diagnostic capabilities and the opportunity to perform minimally invasive therapeutic procedures under image guidance. For over a decade, image-guided procedures have been used for the vast majority of tissue biopsy and percutaneous drainage procedures. This article will address a recent advance in interventional radiology which has occurred in the field of oncology.

A potpourri of percutaneous therapies have previously been utilized for direct intratumoral injection. Chemical agents, such as absolute ethanol, and thermally-mediated techniques, such as radiofrequency ablation (RFA), interstitial laser photocoagulation, microwave therapy or cryotherapy are presently utilized for tumor distribution. All these techniques induce cell death by coagulative necrosis. One of the most exciting techniques for percutaneous tumor destruction is radiofrequency ablation (RFA) and this will be the subject of this overview. This technique has been utilized in the Brown University/Rhode Island Hospital Diagnostic Imaging Department for one year with promising results.

Abbreviations Used:

CT	computed tomography
MRI	magnetic resonance imaging
RFA	radiofrequency ablation

RFA is a technique whereby an alternating current of high frequency radiowaves is emitted from the tip of an electrode or needle placed directly into tissues. The alternating current causes the local ions to vibrate thus creating heat to the point of desiccation (thermal coagulation). (Figure 1). This technique was pioneered in 1920 by Harvey Cushing for the creation of small lesions within the central nervous system. Since that time the technique has been refined such that precise control of lesion size can be achieved by measuring the local temperature and electrical resistance within the tissues being treated. Ablating neural tissue with RF has been successfully used for the treatment of pain from trigeminal neuralgia,[1] facet osteoarthritis and failed back syndrome. Similarly, RF ablation's ability to create localized necrotic lesions indicates why it is well established as the treatment of choice for many symptomatic cardiac arrhythmias[2] and small focal benign painful bone lesions

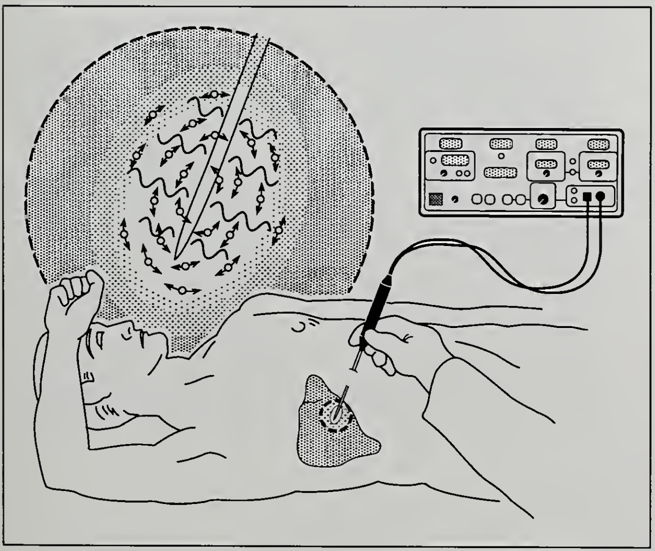


Figure 1. Principle of radiofrequency ablation. A radiofrequency ablation needle is placed into the tumor under ultrasound or CT guidance The emitted radiowaves cause local heating and thermocoagulation of the tissue.

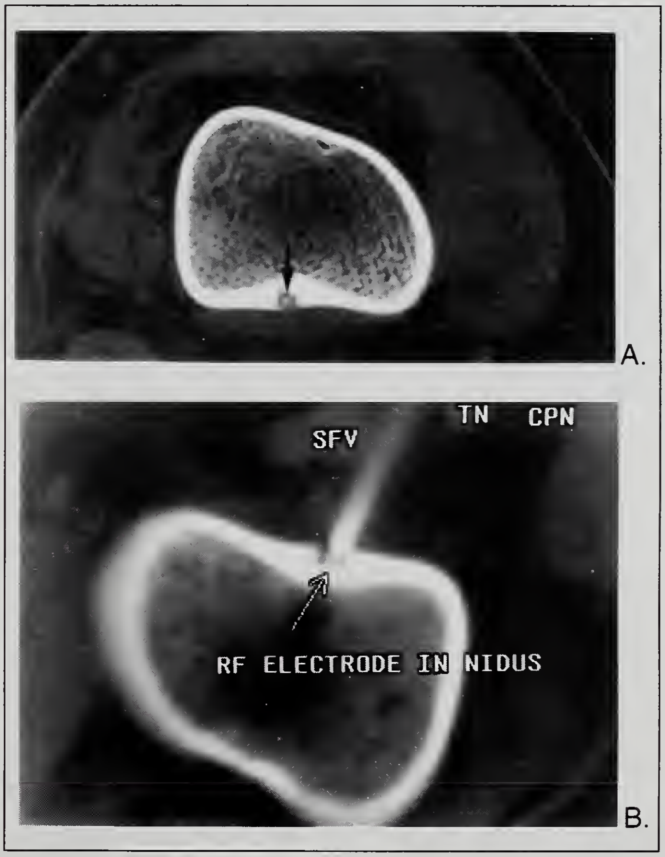
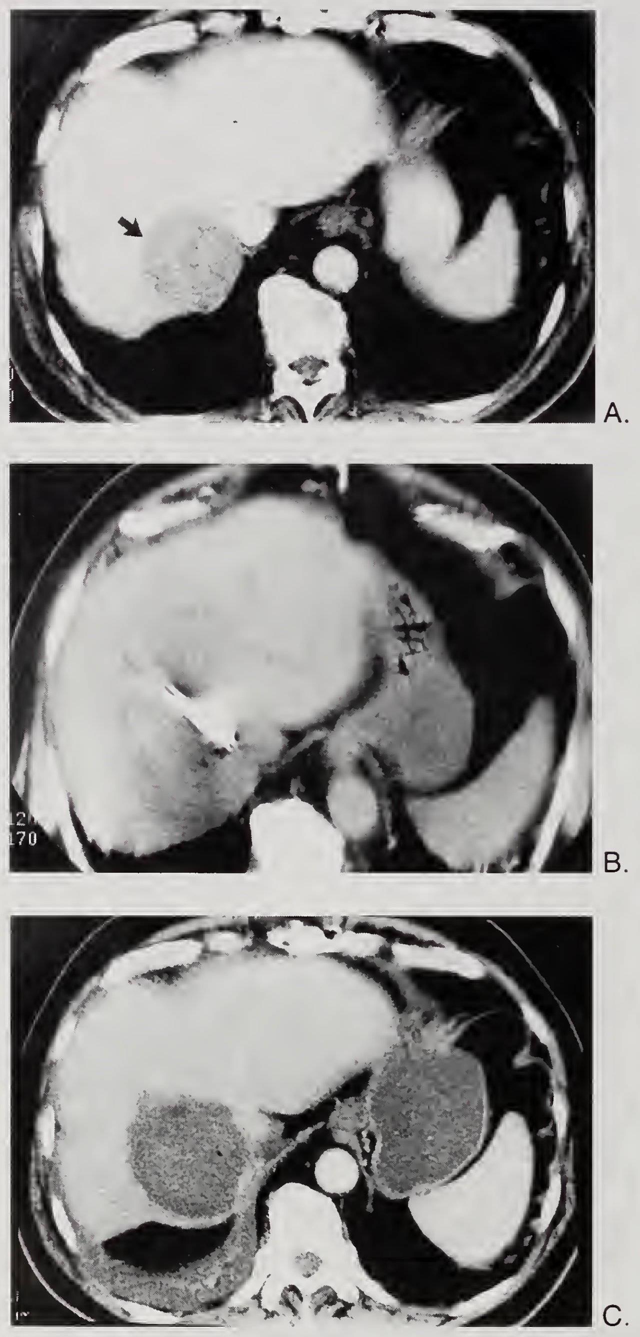


Figure 2. An 18 year old girl presented with local nocturnal bone pain. a. Transverse CT image of the distal femur shows a small cortical bone tumor corresponding to an osteoid osteoma. b. CT image with the RF electrode in place. The nidus of the osteoid osteoma was heated to 90° C for 6 minutes. The patients pain resolved within 24 hours and avoided surgery.



Figrue 3. An 84 year old man shows a 6 cm biopsy proven hepatocellular carcinoma in the dome of the liver abutting the inferior vena cava and hepatic veins. a. Pretreatment CT scan of the liver. b. RF ablation was perfomed with a trident array RF electrode under CT guidance. Four heat lesions were created using two tandem treatments on each side of the lesion. Note the peripheral positioning of the electrode to maximize the ablation volume. c. Two week post-treatment arterial phase spiral CT scan shows complete coagulation necrosis of the mass with no residual enhancement to suggest residual macroscopic disease.

called osteoid osteomas.[3] (Figure 2)

Until recently, a major limitation of RFA was the small lesion size created by this technique. Within the past two years technical advances in RF systems have improved such that heat lesions as large as 5 cm in diameter can be created with a single treatment. This ability makes RFA well suited for the treatment of primary and secondary liver malignancies using a probe small enough for percutaneous place-

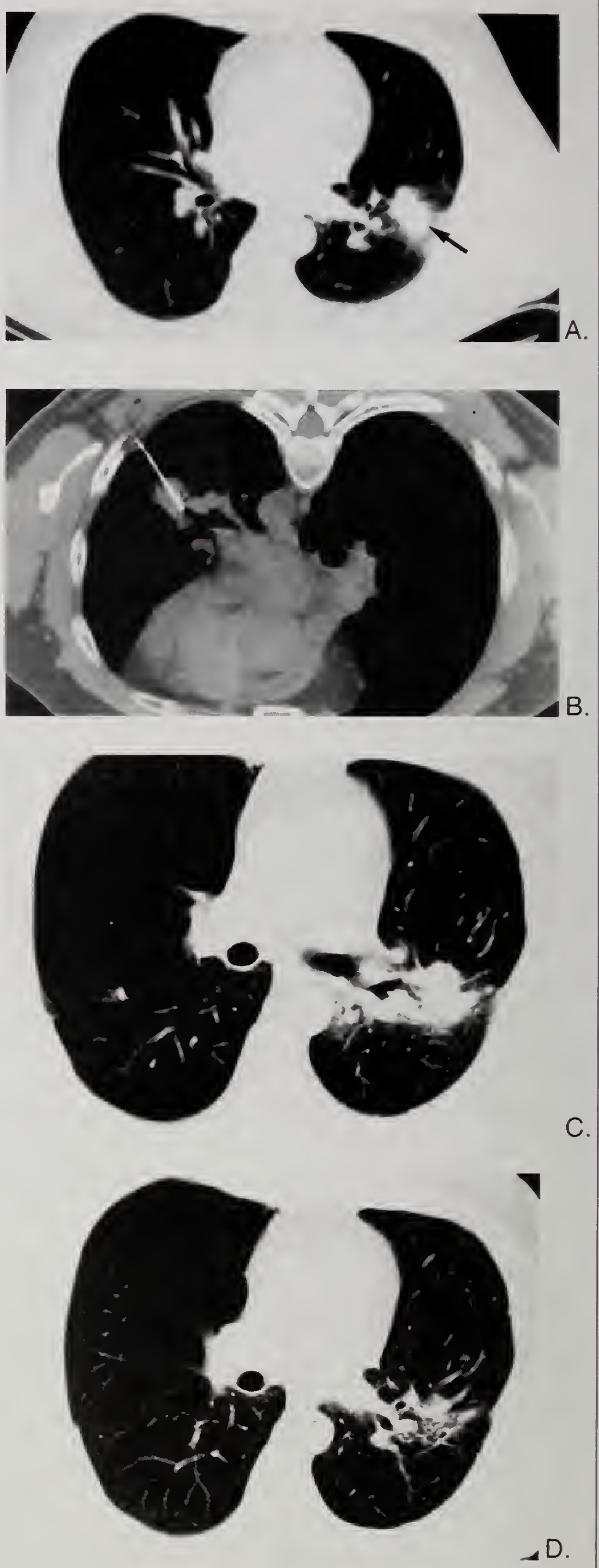


Figure 4. A 45 year old man who had inoperable lung cancer presented with biopsy proven local recurrence in the radiation field after an initial response from chemoradiation. a. A CT of the chest demonstrated local recurrence (arrow). b. A CT-guided RF ablation was performed showing the position of the electrode in the mass. c. A CT immediately following the treatment shows the heat lesion created (arrow). Note the absence of a pneumothorax.

ment on an outpatient basis. For the treatment of hepatocellular carcinomas, RFA has achieved complete necrosis in over 85-90% of cases in lesions smaller than 5 cm with a less than 10% local recurrence rate.[4] (Figure 3) In the treatment of liver metastases, whose very poor responsiveness to percutaneous ethanol injection and other local and systemic chemotherapy is extensively reported in the literature, RFA has a 65-75% reported complete efficacy in the control of local tumor growth in lesions not exceeding 4 cm.[5] Initial reported success with RFA in liver tumors is also coupled with its very low complication rate (<2%).

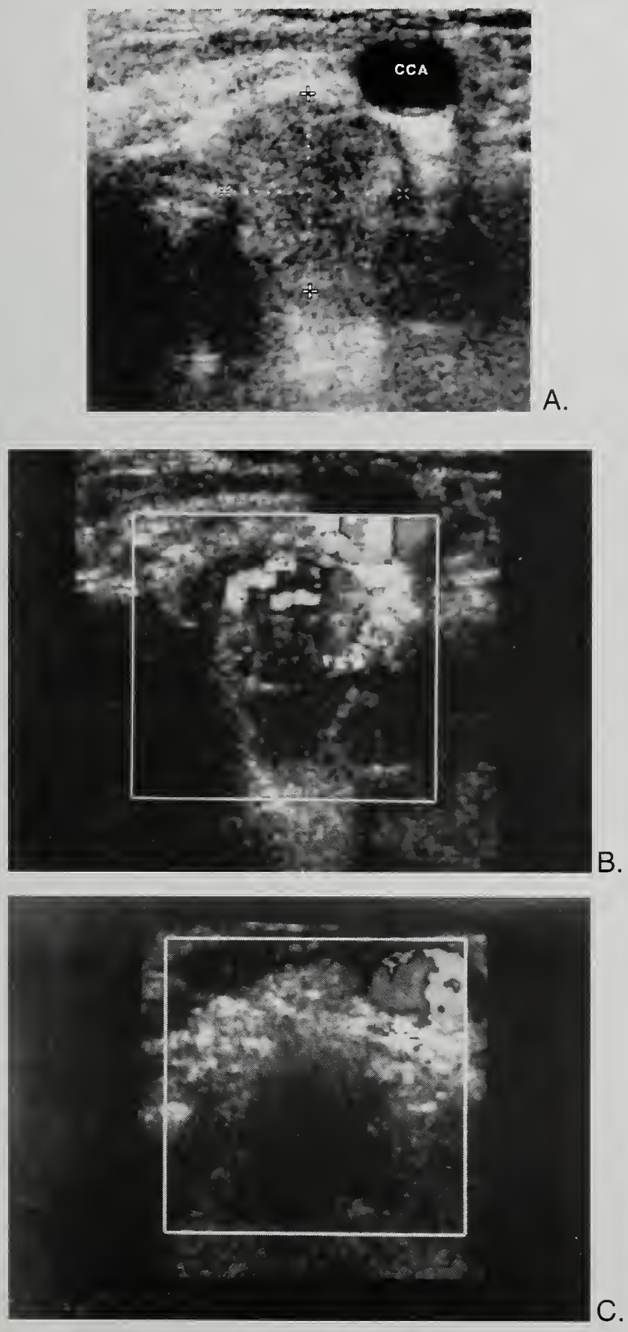


Figure 5. An 84 year old woman presents with recurrent papillary thyroid carcinoma. a. A transverse ultrasound image of the right supraclavicular fossa shows the presence of a 2cm hypervascular mass which was biopsied and proven to be recurrent papillary carcinoma of the thyroid. b. The mass was treated with RFA and a repeat ultrasound shows the lack of color flow within the mass consistent with coagulation necrosis.

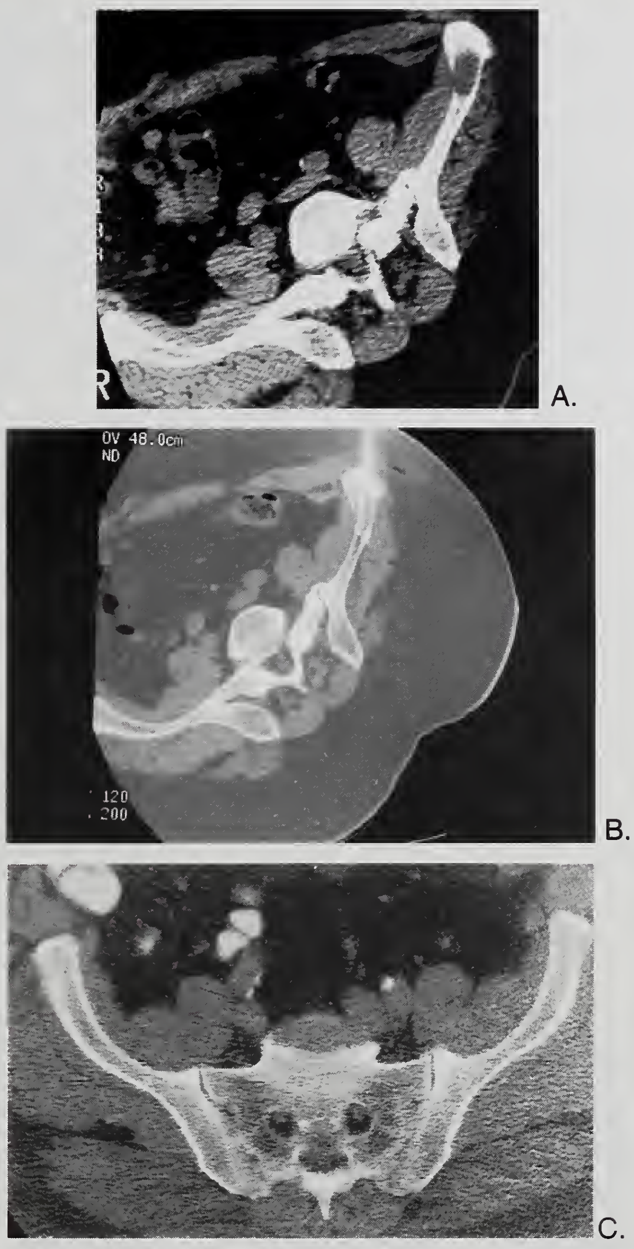


Figure 6. A patient with a history of renal cell carcinoma presents with left hip pain. a. Pretreatment axial CT scan image demonstrates a lytic osseous metastasis to the left iliac crest. b. Percutaneously placed radiofrequency probe within the lesion. c. Follow-up CT scan obtained 14 months later shows complete healing.

Almost all reported complications in liver tumor ablation are focal pain, pleural effusion and regional hemorrhage most if not all requiring no surgical intervention. The treated tissue dies immediately from the thermal coagulation. Over time, thermally coagulated tissue will become resorbed by the body. No evidence of infection within the necrotic masses have been observed and no significant peritonitis has been observed. The goal of the local tumor treatment is to ablate the tumor mass in addition to some adjacent normal parenchyma. Some additional areas in which our preliminary clinical experience with RFA holds promise are the treatment of lung cancer,[6,7] (Figure 4) recurrent head and neck tumors (Figure 5), painful bone metastases[8] (Figure 6) and local control of recurrent retroperitoneal and intraabdominal malignancies (Figure 7). Many patients may not be surgical candidates for tumor removal either due to location and extent of tumor, concomitant debilitating medical conditions or a history of multiple prior surgeries.

Patients are safely treated with RFA almost exclusively in the outpatient setting utilizing local anesthesia and intravenous conscious sedation. RFA can target areas of the body where external beam radiation therapy has been previously ineffective at tumor control or in conjunction with radiation therapy to provide the theoretical advantage of greater up-front tumor cytoreduction.

As new treatment data is produced regarding RFA and other minimally invasive techniques clinical oncologists will have new tools in their armamentarium for the treatment of cancer. This exciting new technique has already demonstrated significant benefits to cancer patients which far outweigh the risk of this minimally invasive procedure.

For the treatment of hepatocellular carcinomas, RFA has achieved complete necrosis in over 85-90% of cases in lesions smaller than 5 cm with a less than 10% local recurrence rate.

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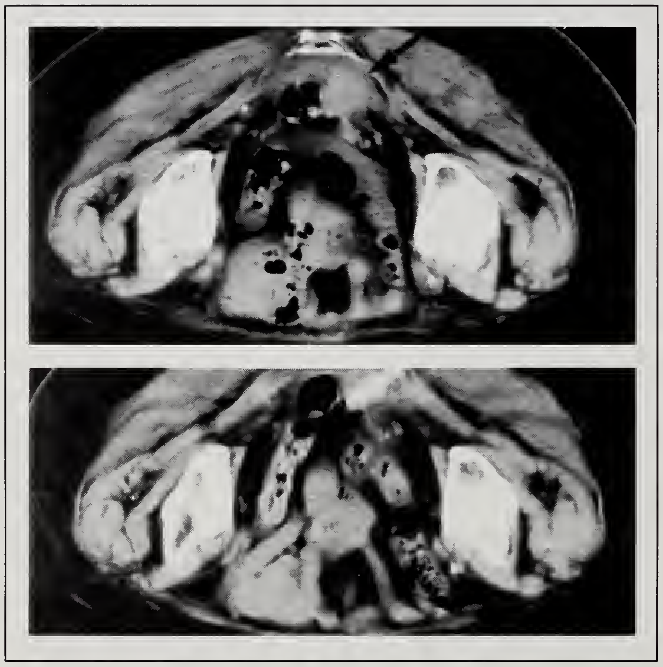

Figure 7. A 76 year old woman with a history of rectal carcinoma presents with posterior pelvic pain. a. A transverse CT image of the pelvis shows a recurrent soft tissue mass adjacent to the sacrum (arrow). The patient had already had two surgeries, external beam radiation and systemic chemotherapy. A biopsy confirmed recurrent disease.b. Under CT guidance the mass was treated with a single RF electrode.

THE CREATIVE CLINICIAN: CASE OF THE MONTH

"The practice of medicine is an art, not a trade; a calling, not a business ..." – WILLIAM OSLER, Aequanimitas

Editor: Anthony Mega, MD; Co-editor: Fred J. Schiffman, MD

From Twisted Ankles

William S. Ferguson, MD

A 7 year old Colombian boy presented to his local hospital Emergency Room with progressive joint pain. He had been well until 4 months before, when he twisted his ankles while playing. Plain X-rays of both ankles were negative. One month later he experienced an injury to his left wrist; an X-ray showed a fracture and his wrist was casted for several weeks. However, his joint pain progressed and within 2 months he was unable to walk. Despite multiple physician visits no diagnosis was made. Psychiatric evaluation was obtained because of his lack of objective physical findings, but did not alleviate his reported symptoms.

Three weeks prior to presentation, he developed a fever to 39° C., which responded to acetaminophen. He was brought to his local hospital, where a cbc showed mild leukopenia and a mild normocytic anemia. Ten days later, his pain markedly increased in severity, accompanied by erythema and swelling of his left ankle and left wrist. A provisional diagnosis of a rheumatologic disorder was made, at which time the family decided to come to the United States for evaluation and treatment.

Initial examination showed an ill-appearing, uncomfortable boy who was unable to stand. His temperature was 39.2° C., pulse 119, respirations 28, and blood pressure 100/56. His left wrist and left ankle were swollen, red, warm, and tender. His right ankle and thigh were tender. He had no adenopathy or hepatosplenomegaly, and no skin rashes; his lung, cardiac, and neurologic exams were normal.

Initial blood tests showed: Hemoglobin 8.2 gm/dl, Hematocrit 23.1%, platelet count 116,000, and wbc 1.7 with 12% polys, 2% bands, 84% lymphs, and 2% monos. His ESR was 113 mm/hr, LDH 401 (normal = 110-210), and AST was 65 (normal = 10-40). His ANA was positive at 1:16; his serum uric acid was 5.0. The rest of his electrolytes and liver function tests were normal. X-rays showed osteopenia of his wrist and ankles.

Because of his pancytopenia, a bone marrow aspiration and biopsy were performed, which showed a decreased cellularity of 20%, with an absence of normal erythroid and myeloid precursors, and no visible megakaryocytes. Approximately 60% of the cells present were L1 lymphoblasts, with the remainder being L2 lymphoblasts. Flow cytometry showed that the blasts were positive for TdT and the cALLa antigen, and negative for myeloperoxidase or surface immunglobulin. These findings were consistent with a diagnosis of B-precursor acute lymphoblastic leukemia

Following initiation of alkaline hydration and oral allopurinol, induction therapy was started with intravenous vincristine, oral prednisone, and intramuscular L-Asparaginase. A spinal tap showed no CNS involvement, and prophylactic intrathecal chemotherapy was administered. The pain and swelling in his joints diminished rapidly; within 4 weeks he was free of pain, and able to ambulate with the help of a walker. One month after starting therapy, his cbc showed: Hemoglobin 8.3, Hematocrit 25.3, platelets 266,000, wbc 6.8 with 44% polys, 1% bands, 47% lymphs, 7% monos, 1% eos. A bone marrow aspiration showed complete re-

Abbreviations Used:

ALL	acute lymphoblastic leukemia
ANA	anti-neutrophil antibody
ESR	erythrocyte sedimentation rate
CNS	central nervous system
JRA	juvenile rheumatoid arthritis
LDH	lactate dehydrate emase
wbc	white blood count

TABLE 1
CLINICAL AND LABORATORY FEATURES OF CHILDHOOD ALL

FINDINGS	% of patients
Laboratory Features	
Leukocyte count (mm3)	
<10,000	53
10,000-49,000	30
>50,000	17
Hemoglobin (gm/dl)	
<7.0	43
7.0-11.0	45
>11.0	12
Platelet count (mm3)	
<20,000	28
20,000-99,000	47
>100,000	25
Symptoms and physical findings	
Fever	61
Bleeding (e.g., petechiae or purpura)	48
Bone pain	23
Lymphadenopathy	50
Hepatosplenomegaly	68

(adapted from reference 1, page 425)

mission: normal cellularity with normal myeloid precursors, megaloblastic erythroid precursors (a common finding with regenerating marrows), plentiful megakaryocytes, and <1% lymphoblasts.

After completing 6 months of consolidation therapy with high-dose methotrexate, he was free of pain and walking normally, and had a normal physical exam. He then received 2 years of maintenance therapy with daily oral mercaptopurine and weekly intramuscular methotrexate, and periodic intrathecal chemotherapy with methotrexate, hydrocortisone, and cytosine arabinoside as CNS prophylaxis. He tolerated his therapy without significant complications. At his last follow-up visit, approximately 3 years following the completion of his therapy, he remains well; his growth has been normal and he is progressingly normally through puberty; his physical exam and blood counts remain completely within normal limits.

Discussion

Acute lymphoblastic leukemia (ALL) is the most common malignancy in children, comprising about 1/4 of childhood cancers (and 80-85% of childhood leukemias) and occurring with an annual incidence of 1/20,000 in children under 15 years of age. The peak incidence is among pre-schoolers 2-5 years of age; there is a slight male predominance, and whites are slightly more likely than blacks to develop ALL.[1]

A diagnosis of ALL is straightforward when presented with the archtypical case of a child with anemia, thrombocytopenia, and leucocytosis with neutropenia and peripheral blasts. However, as Table 1 shows, a significant proportion of children will lack each of these features at the time of diagnosis. In particular, a large proportion of children will have a low or normal wbc, and many will not have identifiable blasts on the peripheral smear.

Common non-hematologic manifestations of acute leukemia include lymphadenopathy and hepatosplenomegaly. Although soft-tissue masses are less common in ALL than AML, they can occur, especially in the testes. Leukemic infiltration of the kidneys is common but rarely detectable on physical examination, and usually does not compromise renal function. CNS involvement is unusual at diagnosis (~3%), but may present either as diffuse meningeal leukemia or focal involvement of a cranial nerve.

Musculo-skeletal symptoms are common but they are usually not the most bothersome manifestations. The most common complaints are of either diffuse aches or knee pain;[2] this is felt to be secondary to pressure resulting from replacement of the marrow space with rapidly proliferating blasts. X-ray studies will often show osteopenia and so-called "leukemic lines," although these findings are not pathognomonic. Focal symptoms in other parts of the skeleton can occur, often with striking X-ray changes, which may precede the development of hematologic changes by weeks or months.[3]

Historically, arthritis was eventually seen in 10-15% of adults with acute leukemia, although this finding is less common now in the era of early treatment. True arthritis is even less common in childhood leukemia.[4] In this case, the child's physicians in Colombia considered a diagnosis of pauciarticular juvenile arthritis, a not unreasonable choice given the constellation of multiple involved joints, fever, malaise, and elevated ESR, and the lack of other physical findings (such as organomegaly or lymphadenopathy) to suggest a lymphoid malignancy. Although leukocytosis is more common than leukopenia in JRA, arthritis and various cytopenias can be seen in other collagen vascular diseases such as systemic lupus.

Most children with acute leukemia will have hypercellular marrows, often so "packed" with leukemic blasts that aspiration can be difficult if not impossible. This child's marrow was atypical in this regard; however, even with a hypocellular marrow, a definitive diagnosis of ALL could be made by the overwhelming predominance of immature lymphoid cells, absence of normal precursors, and marker studies pathognomic for B-precursor ALL. Any lingering doubts concerning the diagnosis would be laid to rest by the child's rapid recovery concurrent with induction chemotherapy

This patient illustrates that leukemia can present with predominately musculoskeletal signs and symptoms, and that leukemia or other marrow infiltrative processes should be kept in mind whenever a child has unexplained or progressive hematologic abnormalities.[5] The diagnosis of acute leukemia can be definitively made (or excluded) only by bone marrow aspiration/biopsy, and this test should certainly be considered in any child with multiple hematologic abnormalities that cannot be completely explained by an underlying illness. The importance of timely diagnosis and treatment is underscored by the fact that with current chemotherapy, children who fall into the low-risk category (age <10 years, initial wbc <50,000, and lack of CNS disease) have a chance of cure that may exceed 80%.[6]

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Rhode Island
Quality Partners, Inc.

Breast Cancer Screening in Rhode Island:
Modest Improvement, Signifcant Challenge

Raymond Maxim, MD

As the current contract cycle begins to wind down and we begin to gear up for the next, we at Rhode Island Quality Partners (RIQP) would like to report on one of our more challenging improvement projects. Last July, I reported to you our plan for improving the breast cancer screening rates for female Medicare beneficiaries. As you may recall, the 1998 project had two major components. A mail campaign was directed at female beneficiaries with the goal of increasing the awareness in breast health among Medicare beneficiaries. It stressed the importance of mammography screening and also reminded them that annual mammography was a covered benefit. The second component was a tool kit sent to primary care physicians in Kent and Providence counties.

The tool kit included a reminder system to prompt ordering of mammograms and information supporting the effectiveness of mammography screening. This included recommendations on breast cancer screening from The American Cancer Society, The U.S. Preventive Services Task Force and The American Geriatric Society.

The overall annual rates improved from an initial 17.9% to 23.7%. In beneficiaries 65-74 years of age, the improvement was 9.2% (from a baseline of 23.2% and improving to 32.4%). This is a significant improvement in a popula-

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Photo: Peter Ventrone

Seated: Dr. Nicholas Iannuccilli: Standing, left to right: Dr. Kathleen Staudinger, Dr. Charles Rosenthal, Dr. Karen Ackerman and Dr. Daniel Druckman: Not pictured are Dr. Paul Hessler and Dr. Rodolfo Zaldivia

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tion of women likely to derive the most benefit from annual mammograms. In slightly older women ages 75-84, the increase is a more modest 6.6%. The evidence for benefit in screening these women is less dramatic but nevertheless important. Weighing the comorbid factors and life expectancy becomes more important in this age group. The group of women older than 84 years also had an increase in mammography screening rates though there is very little evidence regarding the effectiveness of mammograms in this age group.

There was some improvement in reaching the minority population. This is most evident in the black population where the annual rates improved from 12.5% to 17%. This is still far less than rates in the Caucasian community but is still a marked improvement. The screening rates for other minorities improved by a similar percentage. There is a significant challenge for RIQP, and the Peer Review Organization (PRO) program in general, to reduce the racial disparities and still maintain and improve the current performance in the nonminority population. There is clear direction from HCFA to eliminate these disparities among Medicare beneficiaries.

With the above information in mind, RIQP reconvened the Mammography Steering Committee. The committee consists of representatives from physicians, the Rhode Island Department of Health, managed care organizations, hospitals, the Medicare Carrier, and the local community. The committee is currently reviewing the efforts already underway in Rhode Island. There are many organizations, each with their own projects, attempting to raise the awareness of the public and providers to the breast cancer issue. RIQP and the Mammography Steering Committee will host a gathering of these organizations, outreach workers and providers in the fall to share best practices. The goal is to avoid reusing techniques that have been ineffective and concentrate efforts on those that work.

To build on last year's successes, RIQP plans to begin our own efforts. They will include both a beneficiary and provider component. The provider component will likely include providing mammography screening rates for providers. Benchmarking seems to be well received by clinicians. Expectations are that physicians will review their practice patterns to improve their personal rates. In addition, RIQP plans to approach the beneficiaries with a public campaign. This campaign will involve multiple media sources and will include partners from the medical and business community. The goal is to create the demand from the beneficiaries to receive mammograms and other screening benefits. All these efforts depend heavily on the individual physician. The single most important predictor of women receiving a mammogram is that her physician told her that it was important.

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The author assumes full responsibility for the accuracy and completeness of the ideas presented. This article is a direct result of the Health Care Quality Improvement Program initiated by the Health Care Financing Administration, which has encouraged identification of quality improvement projects derived from analysis of patterns of care, and therefore required no special funding on the part of this Contractor. Ideas and contributions to the author concerning experience in engaging with issues presented are welcomed.

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Health by Numbers

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Jay S. Buechner, PhD, and William J. Waters, Jr., PhD

Operating Characteristics of Outpatient Fluoroscopic Systems

Alfred J. Cabral

In Rhode Island, the regulatory oversight of medical diagnostic x-ray facilities is housed in the Department of Health's Office of Occupational and Radiological Health. The state's regulations for the control of radiation contain specific provisions for ensuring radiation safety for fluoroscopic systems. Included in the regulations is a requirement that all fluoroscopic systems be evaluated annually to determine the typical and maximum exposure rates. This report summarizes the results of the most recent survey of non-hospital fluoroscopic systems, covering twenty-one systems located at sixteen facilities. The systems surveyed included eleven systems that were certified by the federal Food and Drug Administration (i.e., manufactured since August 1, 1974) and ten non-certified systems, (i.e., manufactured prior to August 1, 1974).

Abbreviations Used:

FDA	Food and Drug Administration

Methods

The survey protocol included standardized measurements of patient exposure to radiation and image quality during fluoroscopic (continuous) operation and during spot film exposures. A fluoroscopy phantom provided by the FDA was used to represent the average patient during measurement of patient exposure to radiation. Spot film data was collected with the phantom using techniques established

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Table 1. Average Operating Characteristics of Certified and Non-Certified Fluoroscopic Systems, Rhode Island.

Characteristic	Certified Systems	Non-Certified Systems	All Systems
Typical exposure rate	5.2 R/min	4.9 R/min	5.1 R/min
Maximum exposure rate	6.8 R/min	6.1 R/min	6.5 R/min
Spot film exposure	315 mR	382 mR	348 mR
Fluoroscopic image quality			
High contrast items	5.0	2.0	3.6
Low contrast items	5.1	1.7	3.5
Spot film image quality			
High contrast items	5.6	5.2	5.4
Low contrast items	5.3	5.0	5.1

by the facility. A FDA fluoroscopy imaging test tool was used in evaluating image quality in both the typical fluoroscopic mode and under the typical spot film technique. The test tool is a circular device containing eight high contrast evaluators, consisting of meshes in a Plexiglas base varying from 12 to 60 lines per inch, and eight low contrast evaluators, consisting of bored holes in aluminum varying from 0.0063 to 0.068 inches in depth. Accordingly, each system was given a high contrast score and a low contrast score of between 0 and 8 under each method of operation.

Results

All the systems surveyed demonstrated a maximum fluoroscopic exposure rate below the regulatory limit of 10 Roentgens/minute (R/min) that has been established by the FDA for most types of systems. However, substantial variation in the typical exposure rate was seen, with a low of 1.6 R/min and a high of 9.4 R/min. The average typical exposure was calculated at 5.1 R/min, and the average maximum exposure rate was 6.5 R/min. (Table 1) As the normal exposure time in the most common fluoroscopic studies is three to five minutes, patients will typically receive between 15 and 30 Roentgens of exposure during fluoroscopy.

Spot film procedures also showed a wide range of exposure per view with a low of 114 milliRoentgens (mR), a high of 1239 mR and an average of 348 mR. (Table 1) The number and type of spot films taken during a fluoroscopic exam varies, depending on the nature of the study. However, in reviewing records it was seen that the minimum number of views taken was four and the maximum twenty. Therefore, the spot films may add from 1 to 20 Roentgens of exposure to the patient.

Image quality in the visual mode scored much lower than in the permanent spot film mode. The average for visual evaluation was 3.6 high contrast items and 3.5 low contrast items. (Table 1) On several of the non-certified systems, none of the low or high contrast items could be discerned; their average for visual evaluation was 2.0 high contrast items and 1.7 low contrast items. For certified systems, the comparable averages were 5.0 high contrast items and 5.1 low contrast items. On spot films the averages increased to 5.4 high contrast items and 5.1 low contrast items, with no substantial differences between certified and non-certified systems.

Discussion

Fluoroscopic procedures, which normally include x-ray exposure from both fluoroscopy and spot films, deliver a considerable exposure to the patient in comparison to other types of medical radiography. Although the regulatory requirement of a maximum exposure rate of 10 R/m is being complied with, the systems surveyed demonstrated a wide variation in exposure rates. This could be a function of the brightness of the image desired by the radiologist and the sensitivity of the image intensifier system. The non-certified systems, most of which use mirror optics for image intensification, scored poorly in both low and high contrast image evaluation during fluoroscopic mode in comparison to the certified systems, all of which use television optics. This could result in additional exposure time to the patient and an increase in the number of spot films taken. Both would increase the radiation exposure to the patient. Current regulations do not include requirements for visual image quality or prohibit the use of non-certified mirror optics systems. An expeditious means of reducing patient exposure is to minimize fluoroscopic time and the number of spot films taken. Operators of fluoroscopic equipment need to be cognizant of the quantity of radiation exposure delivered to patients during all fluoroscopic procedures. This outreach survey is believed to have achieved the goal of educating operators concerning patients' exposure to radiation during fluoroscopic procedures.

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PUBLIC HEALTH BRIEFING

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

Hepatitis C Virus (HCV): A Silent Epidemic

Utpala Bandy, MD, MPH

HCV is a blood borne viral infection involving the liver (very similar to Hepatitis B) that leads over time to advanced liver disease, liver failure, liver cancer, and death. In fact, it is the leading cause of liver transplantation in the United States.[1] The virus is transmitted through blood or body fluid exchange between persons, such as through blood transfusions, transplants, hemodialysis, needle stick injuries, injecting and nasal inhaled drug use, and through blood entering open skin wounds, and rarely from a mother to infant in the uterus. Many people with HCV have no known risk for infection. A small proportion of cases are sexually transmitted.

Significantly, most persons infected with HCV are not aware that they have been infected. In a large majority of cases, HCV is a chronic, silent disease without overt symptoms, progressing silently and slowly to cause liver damage, often over a period of from ten to 30 years.

HCV was determined to be the primary cause of non-A, non-B hepatitis in 1988.[1] Since then, highly sensitive and specific third-generation ELISA tests (antibody), as well as qualitative and quantitative HCV RNA tests have evolved. Today, these tests are used to protect the blood supply and to screen high-risk individuals for the disease. Early treatment with interferon and/or ribavirin has been demonstrated to be effective in reducing liver damage in selected patients. Seventy percent of U.S. cases are infected with genotype I, a genotype known to respond less well than other genotypes to antiviral treatment regimens.

DISEASE BURDEN: U.S.

Nationally, the Centers for Disease Control and Prevention (CDC) estimates that four million Americans are infected with HCV, and 8000-10,000 persons die from the consequences of this disease every year. HCV causes about 40% of all chronic liver disease in the United States, and costs the nation an estimated $600 million in medical costs annually.[1]

HCV infects Americans regardless of age, race, ethnicity, or socioeconomic status. The prevalence of reported disease is higher in those racial and ethnic groups whose members are at higher risk of disease. The majority of reported cases are found between the ages of 25 and 50, although the disease has been reported at all ages, from newborns to the very old.

Abbreviations Used:

CDC	Centers for Disease Control and Prevention
DOH	Department of Health

As many as 300,000 Americans may have acquired HCV from blood transfusions before 1992, when good screening tests became available. The federal government will soon require all blood banks to undertake a targeted program of notifying persons who may have received HCV infected blood to be screened for the disease.

DISEASE BURDEN: R.I.

The Rhode Island Department of Health (DOH) estimates that 17,000 Rhode Islanders may be infected with HCV. On the basis of national statistics, between 20 and 30% of these people are believed to have had jaundice in the acute phase and therefore would have come under a physician's care for hepatitis. The rest are believed to have been either asymptomatic during the acute phase, or

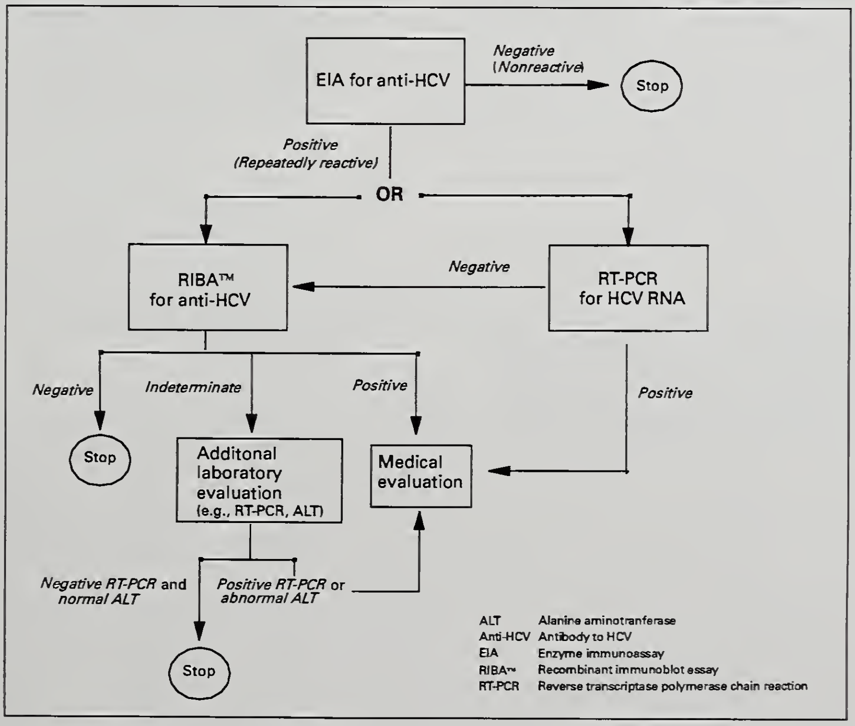
EIA for anti-HCV
Negative (Nonreactive)
Stop
Positive (Repeatedly reactive)
OR
RIBA™ for anti-HCV
Negative
RT-PCR for HCV RNA
Negative
Indeterminate
Positive
Positive
Stop
Additonal laboratory evaluation (e.g., RT-PCR, ALT)
Medical evaluation
Negative RT-PCR and normal ALT
Positive RT-PCR or abnormal ALT
Stop
ALT Alanine aminotranferase
Anti-HCV Antibody to HCV
EIA Enzyme immunoassay
RIBA™ Recombinant immunoblot essay
RT-PCR Reverse transcriptase polymerase chain reaction

Figure 1. Hepatitis C virus (HCV)-infection-testing algorithm for asymptomatic persons

to have had symptoms non-specific for HCV. Most of these people have not undergone screening and do not know they have the disease. Unfortunately, this is just the beginning of the story.

Of the 17,000 Rhode Islanders believed to have HCV, a maximum of 20% will become virus free. The rest (about 13,500) will continue to be chronically infected. Over the next 30 years, between 60 and 70% of these (about 9000 people) will develop liver disease, and some of these will proceed to cirrhosis (about 1500 people), or hepatocellular carcinoma (about 250 people).

PREVENTING HCV

There is little potential that a vaccine will become available any time soon for the prevention of HCV. Therefore, primary prevention focuses on protecting the blood supply, reducing high-risk behaviors such as needle-sharing and high-risk sexual practices, and protecting health care workers from exposure to potentially infected materials. DOH is working to develop a surveillance, prevention, and control infrastructure for the State.

SCREENING FOR HCV

The CDC recommends that persons in the following groups be tested routinely for HCV:

- Persons who ever injected illegal drugs, including those who injected once or a few times many years ago and do not consider themselves as drug users
- Persons with selected medical conditions, including
    - ➢ Persons who received clotting factor concentrates produced before 1987
    - ➢ Persons who were ever on chronic (long-term) hemodialysis
    - ➢ Persons with persistently abnormal alanine aminotransferase levels
- Prior recipients of transfusions or organ transplants, including
    - ➢ Persons who were notified that they received blood from a donor who later tested positive for HCV infection
    - ➢ Persons who received a transfusion of blood or blood components before July 1992
    - ➢ Persons who received an organ transplant before July 1992
- Healthcare, emergency medical, and public safety workers after needle sticks, sharps, or mucosal exposures to HCV-positive blood
- Children born to HCV-positive women[1]

TESTING FOR HCV INFECTION

The CDC recommends that testing for HCV infection follow the algorithm in Figure 1.[1]

Additional information on this subject is available from DOH by telephone (222-2577) or on the web (http://www.health.state.ri.us/disprev/communicable/hepa_c.htm)

Additional information and continuing medical educaton credit for U.S. physicians is available from the CDC web site: (http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/00055154.htm)

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Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	June 1998	12 Months Ending with June 1998		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	232	3,147	317.8	4,005.0
Malignant Neoplasms	199	2,502	252.7	6,826.0**
Cerebrovascular Diseases	48	658	66.4	707.0
Injuries (Accident/Suicide/Homicide)	28	343	34.6	6,695.0***
COPD	33	457	46.2	310.0

Vital Events	Reporting Period	
	December 1998	12 Months Ending with December 1998	
	Number	Number	Rates
Live Births	1,149	13,281	13.4*
Deaths	904	9,710	9.8*
Infant Deaths	(9)	(102)	7.7#
Neonatal deaths	(4)	(77)	5.8#
Marriages	355	7,500	7.6*
Divorces	252	3,156	3.2*
Induced Terminations	342	4,601	346.4#
Spontaneous Fetal Deaths	137	843	63.5#
Under 20 weeks gestation	(132)	(763)	57.5#
20+ weeks gestation	(5)	(80)	6.0#

* Rates per 1,000 estimated population   # Rates per 1,000 live births

**Excludes one death of unknown age

***Excludes two deaths of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 990,225

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

CLINICAL TRIALS DIRECTORY

Medicine & Health/ Rhode Island is pleased to launch this Directory. We intend this to serve as an information clearinghouse for ongoing trials in the state. If you would like to list a trial, please contact: Joan Retsinas, Managing Editor
phone: (401) 272-0422
fax: (401) 272-4946
e-mail: JRetsinas@aol.com.

A Double-Blind Placebo-Controlled Study of St. John's Wort in Depressed Outpatients

Sponsor: Funding is from an unrestricted educational grant from Upsher-Smith Laboratories, Inc.

Purpose: To assess the efficacy, safety, and tolerability of Alterra (Hypericum perforatum)

Principal Investigator: Dr. Scott E. Ewing, Dirctor, Depression and Anxiety Disorders Outpatient Service, McLean Hospital

Patients Recruited: 80 subjects will be recruited, 18-65 years of age, male and female, with no comorbid medical illness, no primary psychiatric diagnosis other than Major Depression, and no history of alcohol or other substance abuse within the past year.

Intervention: Patients have a 50/50 chance of being assigned the active medication Alterra versus a placebo.

Duration of study: 8 weeks

Site: McLean Hospital, Belmont, Massachusetts

Remuneration for patients: No financial remuneration to subjects. All subjects completing the eight-week trial will be offered free samples of Alterra (Hypericum perfooratum) and free follow-up visits for up to two months.

Contact: Mari Andersen, Study Coordinator, phone: (617) 855-2862, fax: (617) 855-3246

Pharmacotherapy of body dysmorphic disorder

Sponsor: National Institutes of Mental Health

Purupose: Evaluate the effectiveness of psychotropic medication for body dysmorphic disorder, a distressing and impairing preoccupation with a nonexistent or slight defect in appearance

Principal Investigator: Katharine A. Phillips, MD

Patients Recruited: Age 18 to 65, both genders, no unstable medical problems, no history of seizures

Intervention: Patients receive a comprehensive psychiatric and medical evaluation. They are treated with medication which appears effective for body dysmorphic disorder. All evaluation and treatment are free of charge. For patients who participate in a placebo-controlled trial, patients randomized to placebo subsequently receive medication at no charge.

Duration of study: Three months

Phase: 4

Site: Butler Hospital

Remuneration for patients: None; however, all evaluations are treatment are provided free of charge.

Contact: Lisa Canuel; phone: (401) 455-6466; fax: (401) 455-6539; e-mail: Katharine_Phillips@brown.edu

Comparison of two marketed antipsychotic medications in the treatment of schizophrenia in elderly subjects

Purpose: The purpose of this trial is to compare two marketed antipsychotic medications for saafety and effiacy on schizophrenia in the geeriatric population.

Principal Investigator: Sumer Verma, MD

Patients recruited: Patients must be greater than 60 years of age and carry a diagnosis of schizophrenia or schizoaffective disorder. They can be male or female. Inpatients, outpatients, nursing home, and boarding care patients may all be eligible for this trial.

Intervention: This double-blind comparison of two antipsychotic medications includes various medical, neurological and cognitive evaluations.

Duration of study: The trial is a total of ten weeks.

Phase: IV

Site(s): McLean Hospital, 115 Mill Street, Belmont, MA 02178

Remuneration for patients: Qualified participants will receive free study-related comprehensive evaluation of their condition. This includes all laboratory tests, and physical and cognitive examinations. Enrolled participants receive 3 months of risperidone and 3 months of follow-up medical care for their condition free of charge, regardless of whether they are able to complete the trial in its entirety.

Contact: Pamela Wheeler, phone (617) 855-3414

A Multi-Center, Placebo-Controlled Trial of Melatonin for Sleep Disturbance in Alzheimer's Disease

Sponsor: Alzheimer's Disease Cooperative Study

Purpose: Brian Ott, MD, is conducting this trial to treat Alzheimer's Disease patients who experience sleep disturbances associated with Alzheimer's Disease. Two doses of melatonin will be used to treat sleep disturbances in order to lessen the burden on caregivers and family members.

Patients Recruited: Anyone 55 years or older with a diagnosis of probable Alzheimer's Disease experiencing sleep disturbances.

Intervention: Two doses of Melatonin vs. placebo

Duration of study: 12 weeks

Phase: III

Site: ALzheimer's Disease & Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860

Contact: Michael Pimental, phone (401) 729-3752

Earlier vs. Later Levodopa in Parkinson's Disease (ELLDOPA)

Sponsor: NIH and the Parkinson Study Group

Purpose: Joseph Friedman, MD, is conducting this trial to compare the effect of early or late treatment of Parkinson's Disease with L-Dopa to answer the question of whether L-Dopa slows or hastens the progression of Parkinson's Disease.

Patients Recruited: Patients must be 30 years or older, must be diagnosed with Parkinson's Disease within the last 2 years. No eldepryl, L-DOPA, amantadine, anticholinergics, antihistamines, antidepressants, or benzodiazepines for previous 30 days.

Intervention: varying doses of Carbidopa/Levodopa vs. placebo

Duration of study: 9 months

Site: Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860

Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

A Phase III Multicenter, Double-Blind, Parallel-Group Placebo Controlled Study of the Effect of Riluzole 50 mg BID or 100 mg BID for Two Years on the Progression of Parkinson's Disease in 1050 Patients

Sponsor: Rhône-Poulenc Rorer

Purpose: The purpose is to treat recently diagnosed patients with the drug Riluzole, in order to study the effect this drug has on delaying the progression of Parkinson's Disease.

Principal Investigator: Joseph H. Friedman, MD

Patients Recruited: Patients must have recently diagnosed Parkinson's Disease with symptoms present for no more than 3 years and must currently be receiving no medications to treat Parkinson's Disease.

Intervention: Two doses of Riluzole or Placebo

Duration of Study: 2 years

Phase: III

Site(s): Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Margaret C. Lannon, RN, MS; phone: (401) 729-3750

Parkinson Disease Collaborative Study of Genetic Linkage (PROGENI)

Sponsor: NIH and the Parkinson Study Group

Purpose: The purpose is to explore genetic, environmental and other factors which may play a role in the development of Parkinson's Disease.

Principal Investigator: Joseph H. Friedman, MD

Patients Recruited: Looking for sibling pairs (brothers and/or sisters) who both have Parkinson's Disease or suspected Parkinson's Disease. Siblings need NOT live in Rhode Island.

Intervention: None

Duration of Study: One visit

Site(s): Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Margaret C. Lannon, RN, MS; phone: (401) 729-3750

Psychosis and Associated Behavioral Disturbances in Patients with Dementia

Sponsor: a major pharmaceutical company

Purpose: The purpose is to determine whether persons with psychosis and behavioral disturbance related to Alzheimer's Disease or vascular dementia benefit from antipsychotic drugs.

Patients Recruited: Patients must at least 40 years old and have hallucinations or delusions associated with dementia caused by Alzheimer's Disease or stroke.

Intervention: One of two antipsychotic drugs, or Placebo

Duration of Study: 28 weeks

Phase: III

Site(s): Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Michael Pimental, phone: (401) 729-3752

A Randomized, Double-Blind Placebo-Controlled Trial to Evaluate the Safety and Efficacy of Vitamin E and Donepezil HC1 (Aricept®) to Delay Clinical Progression to Alzheimer's Disease (AD) from Mild Cognitive Impairment (MCI)

Sponsor: Alzheimer's Disease Cooperative Study and Pfizer/Elsai

Purpose: The purpose is to determine whether treatment of persons with mild forgetfulness can prevent the actual development of Alzheimer's Disease or slow the progression of Alzheimer's Disease

Principal Investigator: Brian R. Ott, MD

Patients Recruited: Patient must be 55 to 90 years of age and have memory difficulties that are not severe enough to interfere with carrying out one's usual daily activities (MMSE=24-30). Must be willing to take only vitamin supplement provided.

Intervention: Multivitamin, Vitamin E, or Donepezil HC1

Duration of Study: 3 years

Phase: III

Site(s): Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Michael Pimental, phone: (401) 729-3752

The Effect of Pramipexole on Depressive Symptoms in Parkinson's Disease Patients

Sponsor: Pharmacia and Upjohn, Inc.

Purpose: To evaluate the effectiveness of pramipexole on the depression and other non-motor symptoms in patients with Parkinson's Disease

Principal Investigators: Joseph H.

Friedman, MD, and Hubert Fernandez, MD

Patients recruited: Men and women with PD, 30 - 85 years old, who have taken no dopamine agonist medications in the past 3 months and are clinically depressed

Intervention: Pramixpexole or placebo

Duration of study: 19 weeks

Phase: IV

Site: Movement Disorders Unit, Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860

Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

Pilot Huntington At Risk Observational Study (PHAROS)

Sponsor: Huntington Study Group (HSG)

Purpose: To observe individuals who are considred "at risk" for developing Huntington's disease (HD) because they have (or had) a parent or sibling with HD. Participants will be examined every 9 months for at least 3 years.

Principal Investigators: Joseph H. Friedman, MD, and Hubert Fernandez, MD

Patients recruited: "At risk" individuals 30 - 54 years old who have never been (and have no desire to be) tested for the HD gene, and who have no definite signs or symptoms of the illness.

Intervention: None

Duration of study: At least 3 years

Site(s): 38 centers in the United States and Canada. Locally at Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860

Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

A Randomized , Double-Blind, Placebo-Controlled Study to Determine the Safety and Efficacy of FK960 in Patients with Mild to Moderate Probable Alzheimer's Disease

Sponsor: Fujisawa Research Institute of America, Inc.

Purpose: The purpose is to assess the safety and efficacy of four different doses of FK960 vs. placebo in patients with mild to moderate probable Alzheimer's Disease and to identify at least one dosage level of FK960 effective in improving symptoms of Alzheimer's Disease

Principal Investigator: Brian R. Ott, MD

Patients recruited: Patients 55 years or older with probable Alzheimer's Disease who are presently not receiving drug treatment for Alzheimer's Disease and who are not presently taking antidepressants or tranquilizers

Intervention: FK960 or placebo

Duration: 6 1/2 months

Phase: II

Site: Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860

Contact: Michael Pimental, phone (401) 729-3752

*** Zyban Collaborative Study***

Sponsor: National Institutes of Health; The Miriam Hospital, a Lifespan partner; and Butler Hospital, a Care New England partner

Purpose The Zyban Collaborative Study is investigating the effects of the combination of Zyban, an FDA-approved medication for smoking cessation, and group counseling to assist with quitting smoking. It is anticipated that this combination of treatments will be particularly effective in the increasing number of "hard core" smokers.

Principal Investigator: Raymond Niaura, PhD, and Richard Brown, PhD

Patients recruited: This free research study is open to cigarette smokers who are at least 18 years old, in good health, smoke 10 or more cigarettes a day, and want to quit smoking.

Intervention: Participants have a 50/50 chance of being assigned the active medication Zyban versus a placebo; they will receive a free medical screening and 12 group counseling sessions.

Duration of study: Participation in the study will last up to one year and will include 12 group visits during a 3-month treatment period, and 3 follow-up visits at 2, 6, and 12 months after the start of treatment.

Phase: Subject recruitment will continue for approximately two-and-a-half years. Participant screenings are scheduled on a continuous basis; new groups begin every seven weeks. Evening groups are available.

Site(s): The Miriam Hospital and Butler Hospital

Remuneration for patients: Participants will receive up to $140 for completing all of the questionnaires and follow-up assessments after treatment ends. Abstinence from smoking is not required in order to be reimbursed.

Contact: The Lifespan Health Connection for further information and screening for participation in the study. phone (401) 444-4800 Dr. Elizabeth Lloyd with specific questions. phone (401) 793-3714, e-mail: ELloyd@Lifespan.org

Moving Forward

Sponsor: National Cancer Institute

Purpose: To examine the feasiblity of cancer survivors adopting a home-based exercise program. To evaluate the effects of home-based modrate exercise on: mood, physical symptoms (e.g., fatigue), weight, sleep, body image and quality of life.

Principal Investigator: Dr. Bernardine Pinto

Patients Recruited: Women ages 18 and over, diagnosed with stage 0-II breast cancer in the past 5 years.

Intervention: A 12-week home-based exercise program

Duration of Study: April 1998 - December 2002.

Site(s): The Miriam Hospital

Remuneration: Non-monetary incentive rewards (e.g., t-shirts)

Contact: Catherine McMahon, phone: (401) 793-3851

THE RHODE ISLAND MEDICAL JOURNAL

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NINETY YEARS AGO

[JUNE, 1909]

Frank B. Fuller, MD, reviewing "Medical Progress During the Past 30 Years," cited benchmarks: Louis Pasteur's germ theory; the waning of diphtheria (he expected 10-20 cases to occur in 1909); hopeful progress in tuberculosis ("we now find that we can recognize the disease much earlier, can cure many, can help all"); the success against rabies (before the Pasteur virus, the mortality if bitten by rabid animals was 15%; afterward, less than 1%); and the cure for hookworm disease. Finally, he described the emergence of antiseptic surgery: "Well do I remember the spectacular entrance of New England's most famous surgeon of his day into the operating amphitheatre, his removal of his immaculate frock coat and the donning of an old blue one that had hung for months, some said for years, conveniently near to the operating table; the succession of operations with perfunctory hand washing between the acts, and finally the replacement of the old blue coat, well bespattered with blood, pus, and discharge of the patients, on its accustomed hook, while the immaculate frock was donned for street ware. Soon Sir Joseph Lister appeared in the same arena, with his steam atomizer, shrouding the field of operation, the hands of the operator, and the instruments in carbolic acid spray."

Charles A. Cooke, MD, in "Appendicitis Complicating Pregnancy," reported on 4 cases: 3 patients recovered, but Cooke cautioned that symptoms were misleading, and diagnosis not easy.

William H. Jordan, in "Infant Feeding," declared, "...there is only one rational food for the baby." For at least the first 10 months, babies should drink natural mother's milk. He called artificial feeding a "necessary evil."

FIFTY YEARS AGO

[JUNE, 1949]

In the summer of 1948, in Munich, when Drs. Alex Burgess, Sr., and Peter Pineo Chase, of Providence, were teaching a course for Displaced Physicians, they met Ivan Basylewicz, MD, formerly Professor of Medicine, Kiev, Ukraine. Before World War II, Dr. Basylewicz had investigated 38 nonagenarians and 34 centenarians. The student-professor showed his American instructors an unpublished manuscript based on his research, and Dr. Burgess adapted one chapter, "Centenarians. The Syndrome of Normal Senility," for publication in this journal. Briefly, Dr. Basylewicz found these villagers and farmers surprisingly young-looking, vigorous, and alert. Only 9 were definitely ill; only 3 were "in a pitiful state of senile marasmus." They had lived austere lives, "characterized by continuous physical work." Most were "passionate smokers, not given to refusing alochol." None were vegetarians. All had married; most reported long-lived ancestors. For the author, this group represented normal aging, unmodified by disease. He found the "unavoidable consequences of continuous [bodily]wear and tear;" but while all organs decreased in activity, "all [were] quite sufficient to satisfy the diminished requirements of the aged body." Signs of normal aging included parchment-like skin, decreased height and weight, muscular atrophy, thin gray hair, wrinkles, and relatively large noses and ears. He found no obvious signs of clinical arterio-sclerosis. Indeed, he discounted Cicero's *senectus ipse morbus* as incorrect: "senility may be unburdened by any pathological changes."

Joseph C. O'Connell, MD, in "The Cost of Medical Care," traced soaring costs to higher utilization (Rhode Island Hospital x-ray department exposed 43,111 films in 1939; 74,110 in 1948. The lab processed 102,527 exams in 1939, 157,902 in 1948. In 1939, 232 patients were referred to occupational therapy; in 1948, 9,034), and to higher hospital charges (one patient/day in all Rhode Island hospitals cost $4.82 to $8.72 in 1939; $9.26 to $17.43 in 1948). After surveying Rhode Island Society members, he reported no dramatic increases in physician fees: in 10 years, an office visit went from $2 to $3, a home visit from $3 to $4. Specialty visits ranged from $5 to $10 both years, with little change. Most surgeons had not raised their fees. Dr. O'Connell also calculated the free care: from October 1947 through September 1948, 6,070 ward patients (staying 91,021 days in Rhode Island hospital) had 4,258 operations. At a cost of $100 per operation, the uncompensated care cost $425,000 - not counting the pre and post-operative care. Similarly, 9,428 patients made 39,119 outpatient visits - at prevailing rates, a subsidy of $157,000.

An editorial ("Blue Cross Participation") praised "the decision of Blue Cross . . . to participate in the Rhode Island Medical Society's prepaid surgical insurance program."

TWENTY FIVE YEARS AGO

[JUNE, 1974]

Stanley M. Aronson, MD, Dean of Medical Affairs, responded to concerns that this new medical school was not educating enough Rhode Island residents. Profiling the Brown University Medical School student body, Dr. Aronson reported that 56 of the 240 students came from Rhode Island, 57 from New York, 30 from Massachusetts. Furthermore, of 44 Rhode Island residents admitted to American medical schools last year, 16 (36%) entered Brown. "Brown medical school is currently host to more Rhode Island residents than all the 22 other New York - New Jersey - New England medical schools combined." As for students' undergraduate degrees, because of the 7 year- continuum, most students received baccalaureates from Brown. Of the 51 non-Brown graduates, no one university predominated (6 graduated from the University of Rhode Island, 3 each from Holy Cross, Harvard, and Columbia).

Francesco Ronchese, MD, in "Malignant Melanoma Turning Non-Malignant-Bleaching Melanotic Freckles," used pictures to demonstrate that, "No matter how numerous and black melanotic freckles may be, they may bleach out spontaneously."

Arnold Porter, MD, in "Blue Shield and HMO's," reported on the 1973 Health Maintenance Organization Assistance Act: it promised to fund 100-150 new HMOs, with $375 million and "almost unlimited" loan guarantees; it wiped out state restrictions on HMOs; and it required all companies employing 25 or more people to offer employees the choice of an HMO if one were available. Currently 22 Blue Cross plans had developed 42 HMO-like projects, with more planned.

This issue celebrated the 25th anniversary of the Veterans Administration Hospital in Davis Park, announcing the good news: The VA Hospital had won approval for a new 2-story ambulatory center; and the Hospital had formally affiliated with the Brown Unversity Medical School.

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Medicine and Health Rhode Island

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Asthma: A CME Issue

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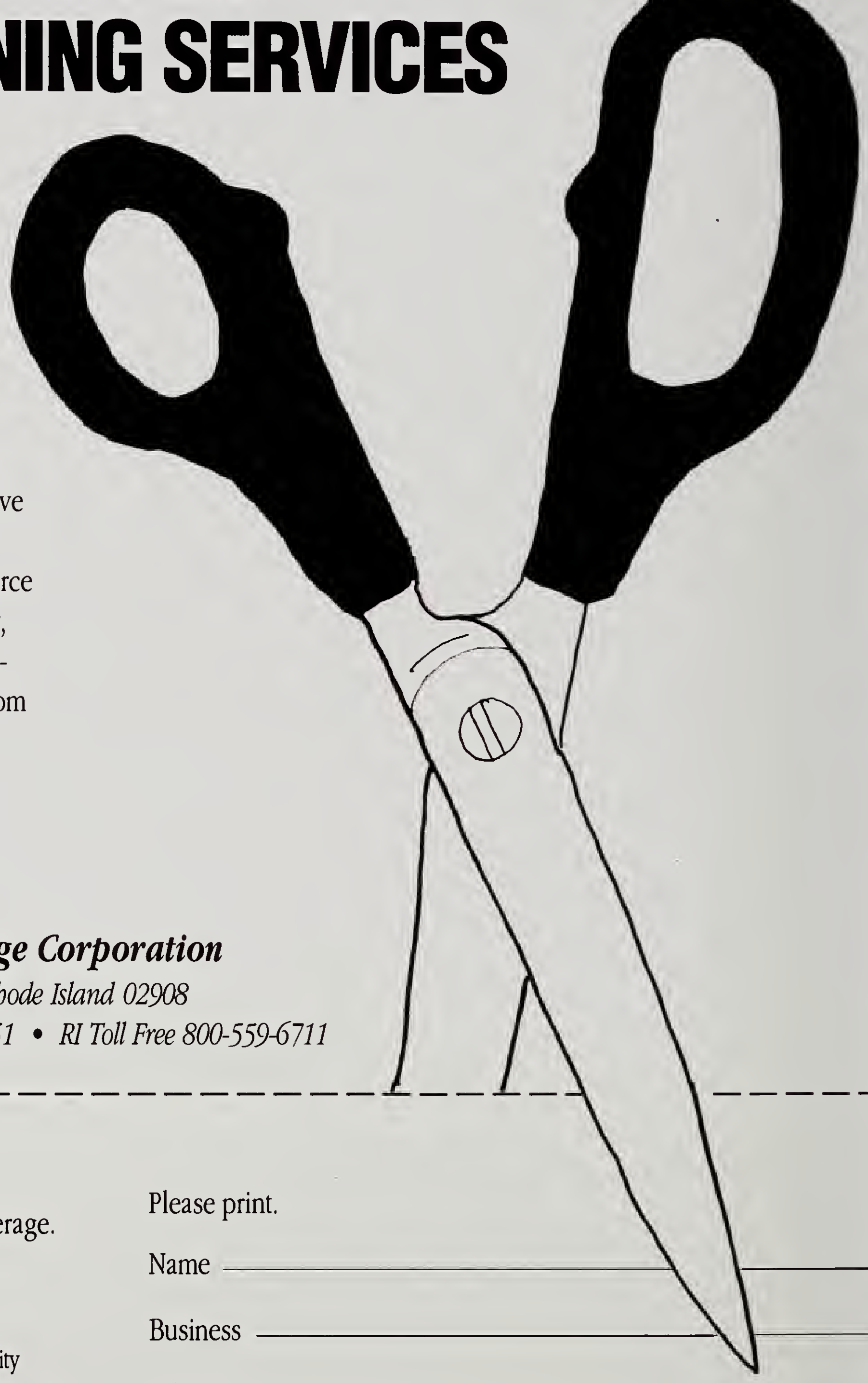

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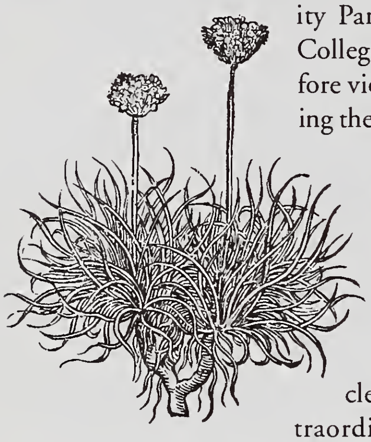
COMMENTARIES

On the Cover: Philip Lieberman's *"TIBETAN SKY"* showcases air unpolluted by industry, by vegetation, by people. In the past, asthmatics sought out "pure" air. Today scientists recognize that air pollution per se does not cause asthma, that the linkage between the offending particles in the air and asthma is more subtle.

Sketches throughout the issue are of the ragweed, grasses, and animals that often trigger asthma attacks.

Winning the Battle but Losing the War: Asthma 1999



This issue of *Medicine & Health/ Rhode Island* devoted to asthma underscores the importance of joint ventures, such as this journal, in bringing together medical and public health issues. This journal is subsidized by the Rhode Island Medical Society, the Department of Health, Brown University, Rhode Island Quality Partners, and the American College of Physicians and therefore views its mission as forwarding the causes of basic and applied medicine, public health and quality care. Asthma is an illness that spans these interests and illustrates the importance of their integration. The articles in this issue make clear that we are in the extraordinary position of understanding asthma better, treating individual cases more effectively, yet finding ourselves overwhelmed by an ever-increasing disease burden. Why this is happening is unclear although many facts are known. Poverty, in some manner, appears to be a major link to this increase but certainly is only part of the explanation. The poor are no poorer now than they used to be and overcrowding is also probably not a whole lot worse than 20-50 years ago. The middle class has seen an increased burden as well. The information in this issue states that air pollution, which would be an easy explanation, is not the answer either.

Whatever the etiology is for this developing crisis, public health, in concert with molecular biology, immune modulation and pulmonary pharmacology, will provide the solution. And, as the case of baby Jose, detailed in Dr. Martin's report, demonstrates, the "ideal" treatment is often not possible for the poor and uneducated, who make up a disproportionate share of the afflicted.

Political will is what we need. If, in this time of unprecedented prosperity, we cannot solve this problem, when will we?

– Joseph H. Friedman, MD

Poverty, the Stepmother of Genius

And old saying declares that the child is father to the adult; that in each child one can detect the rudimentary characteristics and framework of adult accomplishment. Perhaps. But consider the wretched childhood of a boy named for his patron, St. James, and behold then his maturity.

James was born in 1852 in the wretched Aragonese hilltop village of Petilla, a collection of some sixty rude, unadorned houses clinging to the north face of an inhospitable mountain in northern Spain. The only amenity in town was its small church. The annual weather was described as consisting of nine months of winter and three months of hell.

Jim's father was a meagerly educated, itinerant barber-surgeon, a man of indominable spirit, as inflexibly harsh as Petilla's weather. He managed to teach young Jimmy some meager elements of arithmetic and geography. But in his diary he described his son as "a wayward creature, excessively mysterious and unlikeable."

The first 16 years of Jimmy's life consisted of an unending contest of wills between father and son. By age 7 Jim was an accomplished delinquent and a lonely wanderer through the neighboring mountain passes. His life was a succession of vandalism, petty thievery, truancy, beatings and scholastic failures.

In his later writings, Jim recalls only one memorable childhood event: the solar eclipse of 1860. It was not the eclipse itself but rather the astonishing capacity of scientists to have known precisely when this event was to occur. He saw this, in retrospect, as his private epiphany, a greater miracle than all those recorded in his catechism.

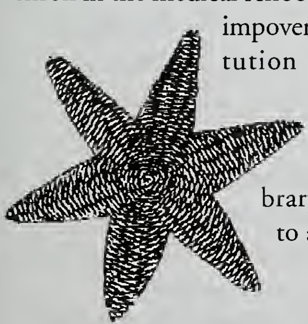
At age 9 Jim was shipped away to a private school for wayward boys. There he was converted from a morbid, inarticulate introvert to a proficient leader of juvenile criminals. His father then brought him to the ancient town of Jaca, to a disciplinary institution with a remorseless motto: "Knowledge enters only with pain." Jim's stay in Jaca was marked by corporal punishment, privation, solitary confinement and periodic starvation. In his isolation, however, he learned to sketch using bits of charcoal and fragments of cigarette paper.

In his eleventh year, Jim managed to construct a cannon, destroying the wall of a neighbor's house. Spain recognized no extenuating circumstances for minor offenders and accordingly Jim was consigned to civil jail, and after a few months shipped away to a reformatory for incorrigibles in Huesca, the city of El Cid. His transfer documents described him as implacably hostile, refractory and antisocial.

Largely through the pleadings of his aggrieved mother, Jim was allowed to be apprenticed to a shoemaker. And while this too proved to be a failure, a chastened adolescent nonetheless managed to attend class for an entire year without any major incident. The Huesca Institute was an authoritarian academy which proclaimed three major evils: rationalism, individual judgment and the writings of Voltaire.

Jim's father had improved his personal station in life. He was now employed by the anatomy professor at Zaragossa University to prepare anatomic dissections. And after all these years of willful conflict, father and son finally discovered some common ground. They jointly labored upon anatomic dissections which Jimmy sketched with exquisite accuracy.

After a year, Jim was permitted to enroll in the medical school. It was an impoverished institution without a single laboratory and a library confined to a corner alcove. Any research was regarded with deep suspicion and the germ theory of disease was summarily rejected.



At age 21 Jim was awarded a licentiate as a physician and surgeon but could find no civilian post. He was then obliged to enter the Spanish army as a field surgeon. The year was 1874 and one of Spain's last colonies, Cuba, was in active revolt and most of the Spanish army had been dispatched to this disease-wracked island. In a decade of attempted pacification, many thousands of Spanish soldiers had died of malaria, yellow fever, dysentery and gunshot wounds. Jimmy labored for two debilitating years as an infantry physician in one jungle encampment after another. He was repeatedly infected with malaria and intestinal parasites. And only after he developed tuberculosis was he finally repatriated to Spain, a malnourished, dispirited veteran, scarcely 24 years old and weighing less than 105 pounds. It required two years in a nursing facility before he could resume work in medicine.

Hardly an auspicious beginning: A refractory, hostile child barely surviving to adulthood; poorly educated in a marginal medical school; divorced from any scholarly activity for four years; profoundly ill, melancholic, periodically belligerent, withdrawn and bereft of social graces; and without job opportunities or professional sponsorship. Jimmy returned to his old job as a laboratory assistant at Zaragossa University investing his entire veteran's pension in the purchase of a new instrument called a microscope. Six years later he accepted a teaching post in Valencia, thence Barcelona and finally, to Madrid where he lived the remainder of his enormously productive life. He died in 1934, beloved by Spain, and honored and cherished by the entire scientific world.

Who was Jimmy? or St. James ? Or, in Spanish, Santiago? He was Don Santiago Felipe Ramon y Cajal, the greatest of Spanish scientists, a man whose professional life had been devoted to a rigorous study of the structure and functioning of the vertebrate nervous system. Through six decades of scientific labor, he had converted the central nervous system from three pounds of mystery to an understandable organ compliant with the rules of general physiology. His research founded the sciences of neurohistology and fundamental neurophysiology. He was the prime teacher of a generation of neuroscientists; and there was not a major city in the Hispanic world that did not have a boulevard or park named in his honor. In 1906 he was awarded the Nobel prize.

Where in Cajal's sociopathic childhood can one find the latent seeds of his genius ? Certainly no one claims that delinquency heralds scientific accomplishment; but adversity in some does result in greater introspection. And just as early poverty may lead to a deeper respect for the material aspects of life, so too a wasted childhood might become a compelling incentive for a lifetime of rigorous study. And perhaps learning to doubt before learning to believe might be an added stimulus to generate new, even heretical, ideas in science.

– Stanley M. Aronson, MD

THE TAPESTRY OF MEDICINE

Stanley M. Aronson, MD, neuropathologist, founding dean of the Brown University School of Medicine, and editor emeritus of *Medicine & Health/Rhode Island*, has been sharing his wit and wisdom with thousands of Rhode Islanders through weekly columns in the *Providence Journal*, and monthly commentaries in *Medicine & Health/Rhode Island*. The just-published book, *The Tapestry of Medicine* (Manisses Communications Group), gathers 73 of those essays. Proceeds will go to the Brown University Medical School. To order a copy, contact the publisher, phone: (800) 333-7771; fax: (401) 861-6370.

Introduction to Special CME Issue: Asthma

We are pleased to welcome you to this special asthma issue of *Medicine & Health/Rhode Island*, the result of a unique collaboration between the Rhode Island Medical Society and the Rhode Island Public Health Association. With hospitalization rates on the rise and a disproportionate disease burden among low-income minority communities, asthma has emerged as a key health challenge for Rhode Island health professionals. In response, the Medical Society and the Public Health Association, champions in their respective fields in advocacy and action for the health of the people of Rhode Island, have pooled their resources and talents for the first time to produce a journal focused on the clinical and public health aspects of asthma.

A special Advisory Board, co-chaired by Dr. Charles Sherman of The Miriam Hospital Pulmonary Division and Dr. Patricia Nolan of the Rhode Island Department of Health, and consisting of health care professionals and community representatives with expertise and experience in asthma, has been instrumental in assisting the journal's Editorial staff in the selection of the topics for this issue. Readers will be challenged to conceptualize asthma not as merely a disease impacting the individual patient, but as a health issue within a broader family, community, and environmental context.

This project has been supported through the *Cooperative Actions for Health Program* (CAHP), a collaborative grant project co-sponsored by the American Public Health Association and the American Medical Association, with funding from The Robert Wood Johnson Foundation. As leaders in CAHP and in the National Medicine/Public Health Initiative, we believe that collaboration between medicine and public health is essential for improving the health of the people we serve, as individuals and as communities. We congratulate the Rhode Island Medical Society and the Rhode Island Public Health Association for this landmark collaborative effort, the first step, we believe, in an on-going relationship in service for the health of the people of Rhode Island.

Reed V. Tuckson, MD
Senior Vice President for Professional Standards
American Medical Association

Mohammad N. Akhter, MD, MPH
Executive Director
American Public Health Association

For CME Credit

Medicine & Health/Rhode Island presents these articles as part of a CME offering. At the end of the articles, you will find information on registration as well as the CME questions.

A CONFERENCE:
POPULATION BASED PRIMARY CARE – From Idea to Health Care System

August 12 – 13, 1999.
W. Alton Jones Conference Center, West Greenwich, Rhode Island.

Sponsored by Rhode Island Academy of Family Physicians.

Population-based primary health care is that system of health care distribution which assigns every person residing in a geographical area to a population-based primary health care practice located in that geographical area, and makes every population-based primary health care practice located in a geographical area responsible for the primary health care of every person residing in that geographical area.

For more information, please contact: Nancy Leggat, phone/fax: (508) 336-5416

Asthma: Profile of an Epidemic

Charles Sherman, MD, MPH

Incidence:		
Age	1980	1993-94
newborn to age 4	42.8/1,000	74.4/1,000
ages 4-14	30.7	53.8
all ages	22.2	57.8
Race		
black	34.0	57.8
white	30.4	50.8
other	22.5	48.6
Mortality by race		
black	4.20	3.85
white	2.66	1.51
other	2.57	1.77
Prevalence:	1975	1993-95
people who went to MD for asthma	4.6 million	10.4 million
	1980	1998
people who self-report with asthma	6.8 million	17.3 million

Cause for Increase: Not definitively known

Hypothesized Causes:

- * Changes in diagnostic reporting: today persistent respiratory distress (pediatrics) and chronic obstructive pulmonary disease (adults) may be more often diagnosed as asthma
- * Genetics: today more people may have genetic predispositions
- * Environmental factors: changes in both indoor and outdoor allergens, irritants, and pollutants have been linked to asthma increases

Triggers/Irritants:

1. allergens, such as cats, dogs, mites, cockroaches, grain, mold, and pollen
2. cigarette smoke
3. respiratory infections
4. exercise
5. fumes from cleaning solvents and other chemicals
6. allergies to specific foods and medications (e.g., aspirin and advil)

Cure: None

Treatment: Managing the Disease

Patients manage the disease by:

- * avoiding triggers where possible
- * monitoring symptoms
- * taking medication as prescribed
- * hyposensitization to specific allergens, if identified

Medications:

*Controller medications:

1. inhaled steroids
2. cromolyn and nedocromil
3. long-acting beta$_2$ agonists
4. leukotriene modifiers (approved by the FDA since 1997)

* Quick relief medications

1. beta$_2$ agonists
2. anti-cholinergics

Environmental:

- * reducing household contaminants ("cleaning" to reduce exposure to asthma is similar to "cleaning" to reduce exposure to lead)

Source of Numbers: Centers for Disease Control

This issue, funded by the Robert Wood Johnson *Cooperative Actions for Health Program* (CAHP) grant, offers Rhode Island clinicians a collection of papers that present asthma from a public health vantage - seeing it not as a single clinical incidence for one patient, but as a population hazard.

Patricia Nolan, Director of the Department of Health and my co-editor, discusses the dilemma of reporting: on the one hand, to document the epidemic, the country's public health departments need consistent uniform data. Today's pastiche from insurers' bills, hospital discharges, school absentee records, and patients' self-reports tracks the rising incidence/prevalence, but gives epidemiologists weak benchmarks. On the other hand, making asthma reportable means setting clear diagnostic criteria, and deciding who will pay for the costs of reporting. Although the Centers for Disease Control has recommended against making asthma reportable, as Patricia Nolan explains, it may be time to reconsider that recommendation.

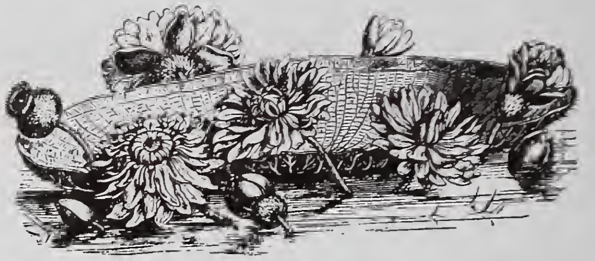
The management of asthma (self-management plan, instructions as to monitoring, medications, environmental triggers) begins in the physician's office. I condense the NIH guidelines (1991 and 1997 editions) into a useful template for physicians who must launch patients into a rigorous regimen of management in only 2 to 3 visits. Primary care physicians, however, confront patients who may believe that their wheezing denotes asthma, but who don't in fact have asthma. Michael Fine discusses the difficulties of diagnosis in primary care practice.

Medications are central to the control of asthma, but we have no clear-cut magic bullets that will work for all patients at all times. Mary Ann Passero, a pulmonologist at Rhode Island Hospital, and David Chronley, a pediatrician in South County, recreate the conversation about inhaled steroids in pediatrics that the spurt of new studies has raised. The leukotriene modifiers are the first new class of drugs approved by the FDA for asthma in 20 years. Cynthia Wedekind, from the University of Rhode Island's College of Pharmacology, discusses this new medication, approved by the FDA for the past 2 years, and now prescribed for the most part for adult patients.

For pediatric patients, the child's pediatrician is a (hopefully) rare figure in the child's life, seen occasionally. The teacher, though, sees the child 180 days (again hopefully) a year; and the school nurse can be an ally, helping the child monitor symptoms, recognize attacks, and take medication. Indeed, the NIH has explicitly recommended a school monitoring plan. Ann Kelsey Thacher and Rosemary Reilly-Chammat, from the Department of Health, describe school policies (for instance, since 1998 children can carry inhalers to school; smoking is barred at all school events, even those off-site), and suggest ways physicians can work with school nurses.

Physicians can also work with community "asthma" organizations. Patients and families will need intensive education (anecdotal evidence suggests that many patients use peak flow meters incorrectly, that they discard inhalers and stop taking their medication, that they overlook key symptoms). Betina Ragless, from the American Lung Association of Rhode Island, lists the educational initiatives happening in the community. And since patients are increasingly prone to seek information, advice, and support in the virtual community, a table lists some asthma-related web sites.

The triggers to attacks are based in the environment. Robert Vanderslice and Lynn Bibeault, from the Health Department, summarize relevant "clean environment" legislation, and describe several local housing initiatives. Since asthma disproportionately affects inner-city, particularly minority children, and since those children often live in substandard rental housing, the campaign to reduce environmental triggers segues into campaigns to improve housing (including the reduction of lead-based paint), to increase poor children's access to consistent health care, to reduce poverty. Bruce Kogan, from Roger Williams Law School, gives insight into the rights, and the dilemmas, of the worker with asthma, under the Americans with Disabilities Act.



For a population, the impact of asthma can be measured in mortality and morbidity statistics. Jay Buechner, from the Health Department, summarizes recent data, which overall show asthma-related mortality declining in the past decade, and asthma-related morbidity remaining stable.

Finally, for an individual, the impact of asthma translates into suffering. Carla Martin, a resident at Hasbro Hospital, presents a case that weaves all the social ills that make management of asthma so difficult: poverty, poor housing, transiency, unstable - if not abusive - family relationships. The toddler, with a genetic history of asthma, has a sibling with recurrent respiratory infections. If this issue ultimately helps those patients, the grant money will be well-used.

ACKNOWLEDGEMENT

This issue was a collaborative endeavor, beginning with the partnership of the Medical Society and the Rhode Island Public Health Association, which jointly received a *Cooperative Actions for Health Program* grant. The Continuing Medical Education advisory committee oversaw the delineation of objectives and the selection of articles. I thank those members: Patricia Nolan, MD, Celia Gomes-McGillivray, MPH, the president of the Rhode Island Public Health Association; Joan Retsinas, PhD, managing editor, *Medicine & Health/Rhode Island,* Barbara Niekerk, MEd, Director of Continuing Medical Education at Brown University School of Medicine, Edward Westrick, MD, from the Rhode Island Quality Partners; William Corwin, MD, from Harvard Pilgrim Health Plan, Stanley Bloch, MD, from the Providence Ambulatory Health Center, Carla Martin, MD, and Ersilina Bencosme, MD, from Rhode Island Hospital, Jeffrey Newell, DPharm, from CVS-Health Care Services, and Toby Liebovitz, a parent-teacher-advocate.

Charles Sherman, MD, MPH, is a an Associate Professor, Brown University School of Medicine, and co-guest-editor of this issue.

Should Childhood Asthma Be Reportable?

Patricia Nolan, MD

Asthma in childhood is an important health problem. It disrupts school and play. It causes illness and even death. It costs a substantial amount in health care dollars. It interferes with our children reaching their full potential. And many of its negative effects can be prevented.

Our public sources of information about morbidity and mortality are *ad hoc* sources: hospital discharge data and death certificates to confirm the worst effects of asthma, anecdotal or case-by-case data to assess the breadth of effects in the population. (Private managed care organizations do track asthma among their enrollees, but don't generally share that data with the public.) The gradation of the severity of asthma reflects clinical concerns, but is hard to translate into incidence and prevalence data. The lack of consistency in definitions of clinical severity, incidence, and prevalence makes it more difficult to identify modifiable factors to reduce asthma morbidity. The same lack of clarity has limited our ability to measure the impact of strategies we initiate.

Would making asthma reportable help us identify modifiable factors and begin to reduce the adverse effects of asthma?

> *Without population-based surveillance, we are working in the shadows, trying to solve a major public health problem whose dimensions we cannot really see.*

Making asthma reportable could lead to agreed-upon criteria of severity for epidemiologic purposes, and that alone is useful. Reporting provides a basis for defining an asthma "event". Reporting is only a starting point, however, and must be supported by investigation and analysis resources. If supported, reporting opens a surveillance system with the capacity to study incidence, prevalence, and modifiable factors over time. Using a common set of criteria, we are able to compare incidence and prevalence across time, among geographic areas, demographic groups (age, race-ethnicity, gender), socio-economic groups, and among states. Local data on severity levels and risk factors can be clarified, and long term prevention goals can be set realistically and then measured.

An alternative to reporting in the "infectious disease" model is registry development, a method of surveillance more commonly used for chronic illnesses such as cancers. The investment in a registry may be larger, because there is more emphasis on tracking individual outcomes over time.

Either a reporting or a registry system would enhance our understanding of childhood asthma and our accountability for interventions. Full value of surveillance requires turning information around and assuring that families and physicians know what the data show. The sharing of data across insurers, providers and even state lines is difficult. A surveillance system at the state level can protect confidentiality and yet share important data.

The Centers for Disease Control and Prevention have not promoted reporting of childhood asthma. They have funded surveillance projects in several states and cities, using emergency room visits as a critical factor. While a useful strategy, emergency room visits are only a part of the spectrum of this important condition, but a good reflection of strategies to reduce emergency room use.

There is interest in establishing a non-governmental registry for asthma, but the potential for competing registries is high.

Health plan disease management protocols may collect the essential information, but they are hindered by the movement of enrollees among plans. Sharing information between plans is hindered by confidentiality and competition concerns. Competing registries and health plans require a central repository to achieve population-based surveillance. Otherwise we cannot tell if we are over-counting or under-counting morbidity.

It will require an investment by state government and health care providers, and insurers, but it is time to make asthma a reportable condition. Without population-based surveillance, we are working in the shadows, trying to solve a major public health problem whose dimensions we cannot really see.

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Office Management of Asthma

Charles Sherman, MD, MPH, and Darlene Arthurs, RN

Asthma is a chronic illness characterized by airway inflammation, partially reversible air flow obstruction and increased airway responsiveness. It affects almost 15 million people in the United States and is the most common chronic illness of childhood.[1,2] Asthma prevalence and incidence have increased over the last 20 years. Individuals with asthma account for more than 100 million days of restricted activity and 470,000 hospitalizations per year. Each year 5,000 people die from their asthma. Blacks, aged 15-24 years, are the most likely to die from their disease.[3] As can be appreciated, the diagnosis and management of asthma are of paramount importance for all health care providers.

To help educate physicians throughout the country, an expert panel convened by the National Institutes of Health produced *Guidelines for the Diagnosis and Management of Asthma* in 1991 [4] and an updated version in 1997.[5] These clinical guidelines emphasized four main components in effective management: objective measures of lung function, environmental control measures, comprehensive pharmacologic therapy, and patient education. The guidelines have provided a wealth of information for physicians caring for asthmatics, whether they be children, adults or the elderly. Despite widespread distribution of the expert panel reports, physicians in practice have found them difficult to apply. Large numbers of outpatient visits and time restraints for patient education have been identified as barriers to implementing the guidelines. Further, the guidelines do not provide a usable format for individual patient visits. This article seeks to provide clinicians with a practical approach to the diagnosis and management of asthma, incorporating the expert panel's guidelines into office practice.

> *Despite widespread distribution of the expert panel reports, physicians in practice have found them difficult to apply.*

What follows is a detailed description of what should occur during the initial office visit and the two follow-up visits, allowing an expedient diagnosis of asthma and optimal management of the disease (Figure 1).

During the first office visit, several important goals need to be achieved. The diagnosis of asthma must be confirmed and the patient needs to be instructed in using a peak flow meter and recording a daily symptom diary. (Patients are given a daily diary to complete). In addition, the patient (and/or parent) must understand their medications: proper dosage, possible side-effects, ramifications of a missed dose.

Because the 30-40 minute visit does not allow time for much discussion, the clinician should give the patient (parent) questionnaires before the visit. (Admittedly, patients with low levels of literacy may gain little, if not be deterred, by written questionnaires; those patients will need additional oral instructions.) (Center insert #1). This questionnaire should not only list a variety of symptoms suggestive of asthma, but include the pattern of symptoms, and precipitating and/or aggravating factors for the symptoms. Questions on the family and social history including items on smoking, home environment and social factors that may interfere with adherence to a management plan must also be included. The symptoms most characteristic of asthma are cough, which is worse at night, recurrent wheezing, recurrent difficulty in breathing and recurrent chest tightness. These symptoms are usually worse in the presence of exercise, viral infection, exposure to animals with furs or feathers, mold, smoke, pollen or changes in the weather. Symptoms that occur at night are particularly worrisome.

If the patient has symptoms suggestive of asthma, then pulmonary function testing is necessary to confirm the diagnosis. Spirometric measurements before and after the patient takes a short-acting bronchodilator must be undertaken. A 15% increase in FEV_1 and FVC and/or a 25% increase in FEF25-75 are supportive of reversible air flow obstruc-

Figure 1:
Goals of Initial and Follow-up Visits for Asthma Management

Initial Visit
1. Confirm Diagnosis
   a. History
   b. Physical Exam
   c. Spirometry
2. Start Appropriate Medications
3. Patient Education
   a. Use of Peak Flow Meter
   b. Completion of Daily Symptoms/ Peak Flow Diary
   c. Medication Effects and Side Effects

1st Follow-up Visit (2 weeks)
1. Review Daily Symptoms/Peak Flow Diary
2. Review Medication Use and Delivery Technique
3. Medication Adjustment
4. Patient Education
   a. Warning Signs of Exacerbation
   b. Trigger Control

2nd Follow-up Visit (6 weeks)
1. Review Daily Symptoms/Peak Flow Diary
2. Review Medication Use and Delivery Technique
3. Medication Adjustment
4. Patient Education
   a. Asthma Management Plan

tion. Peak flow measures, although important in monitoring treatment, are not useful in the initial diagnosis of the disease. Bronchoprovocation with methacholine may be necessary if spirometry is normal or near normal, but the patient has symptoms suggestive of asthma. Adolescents and school-age children may be best tested in a laboratory familiar with evaluating children in this age group. Chest x-rays are rarely necessary other than to exclude other diagnoses.

... the goals of the initial visit are to confirm the diagnosis, to educate the patient (and/or parent) on using the peak flow meter and their medications, and convince the patient (and/or parent) to keep a daily diary.

Several other diseases must be considered. In children, the clinician should rule out upper airway diseases (allergic rhinitis and sinusitis), obstruction involving the large airways (foreign body in the trachea or bronchus), vocal cord dysfunction, or obstruction involving smaller airways (viral bronchiolitis or obliterative bronchiolitis, cystic fibrosis, bronchopulmonary dysplasia, heart disease). In adults, chronic obstructive pulmonary disease, congestive heart failure, pulmonary edema, pulmonary embolus, mechanical obstruction of the airways, pulmonary infiltrates with eosinophilia, cough secondary to drugs, and vocal cord dysfunction may mimic asthma. A careful history is useful in eliminating these other possibilities.

After quickly establishing the diagnosis during the first part of the initial visit, patient education is the next important part of the visit. The patient (and/or parent) must be instructed on using a peak flow meter to assess air flow each day and at different times during the day. The patient (and/or parent) also needs to be educated on how to use appropriate medications with a holding chamber. This information will be recorded on the daily diary over the next two weeks (Center insert #2. The diary information will eventually be used to characterize the patient as having mild intermittent asthma, mild persistent disease, moderate persistent disease, or severe persistent disease (Figure 2). This characterization is important as stepwise therapy is recommended based on the severity of disease (Figure 3).

It is important to emphasize that physical exercise should be encouraged for all asthmatic children.

At the end of the visit, the clinician should give the patient (parent) additional written materials on medications, peak flow meters, and the diary.

Thus, the goals of the initial visit are to confirm the diagnosis, to educate the patient (and/or parent) on using the peak flow meter and the medications, and convince the patient (and/or parent) to keep a daily diary.

A follow-up visit should occur in two weeks. In the waiting room, the patient (and/or parent) should complete a follow-up questionnaire (Center insert 3). At the time of the visit, the clinician should review the daily diary for respiratory symptoms, peak flow and any adverse effects to medications. The patient (and/or parent) should also be questioned about any missed work or school, any reduction in usual activities, and any disturbance in sleep due to asthma. Adherence to the medication regimen, inhaler technique, the frequency of use of each medication, and side effects of medication should be ascertained. Spirometry should be repeated. Based on the patient's reported symptoms and spirometry measures, the clinician may adjust medication. (Center insert 4) The patient (and/or parent) should then be given information on warning signs of asthma episodes, managing trigger control and information on when to stay home and when to go to the Emergency Room.

A third visit should occur in six weeks. Again, the daily diary should be assessed for changes in symptoms. Spirometry should again be performed. Patient (and/or parent) teaching should be reinforced on the proper technique of peak flow and medication usage including the holding chamber. Direct observation of these skills is an excellent method of assessment. Based on spirometry and the respiratory symptoms, the clinician should again adjust medication. (Center-insert #4) In this third visit, formalization of the self-management plan should be done. The patient (and/or parent) should have a good knowledge of his/her symptoms and triggers. The patient (and/or parent) should also understand how to determine when to stay home and when to go to the Emergency Room. The patient (and/or parent) should understand the medications and their side effects. The patient (and/or parent) should demonstrate knowledge of the basic facts about asthma and show skill in using the inhaler holding chamber and the peak flow meter. The patient should have a formal asthma management and action plan at the end of this visit. Adolescents and older school-age children should be directly involved in setting therapeutic goals and devising the individual asthma management plan. A copy of the written asthma management plan should be given to the appropriate school representative. It is important to emphasize that physical exercise should be encouraged for all asthmatic children.

At all subsequent visits, the clinician should reassess the patient's symptoms, peak flows, quality of life and knowledge of disease. The patient (and/or parent) should establish treatment goals; e.g., being free from severe symptoms day and night, having the best possible lung function, being able to participate in any activity of choice, not missing work or school because of asthma symptoms, not needing

FIGURE 2 Stepwise Approach for Managing Asthma in Adults and Children Older Than 5 Years of Age

Goals of Asthma Treatment

- Prevent chronic and troublesome symptoms (e.g., coughing or breathlessness in the night, in the early morning, or after exertion)
- Maintain (near) "normal" pulmonary function
- Maintain normal activity levels (including exercise and other physical activity)
- Prevent recurrent exacerbations of asthma and minimize the need for emergency department visits or hospitalizations
- Provide optimal pharmacotherapy with minimal or no adverse effects
- Meet patients' and families' expectations of and satisfaction with asthma care

	Classify Severity of Asthma		
	Clinical Features Before Treatment*		
	Symptoms**	**Nighttime Symptoms**	**Lung Function**
STEP 4 **Severe Persistent**	■ Continual symptoms ■ Limited physical activity ■ Frequent exacerbations	Frequent	■ FEV_1 or PEF ≤60% predicted ■ PEF variability >30%
STEP 3 **Moderate Persistent**	■ Daily symptoms ■ Daily use of inhaled short-acting $beta_2$-agonist ■ Exacerbations affect activity ■ Exacerbations ≥2 times a week; may last days	>1 time a week	■ FEV_1 or PEF >60% - <80% predicted ■ PEF variability >30%
STEP 2 **Mild Persistent**	■ Symptoms >2 times a week but <1 time a day ■ Exacerbations may affect activity	>2 times a month	■ FEV_1 or PEF ≥80% predicted ■ PEF variability 20-30%
STEP 1 **Mild Intermittent**	■ Symptoms ≤2 times a week ■ Asymptomatic and normal PEF between exacerbations ■ Exacerbations brief (from a few hours to a few days); intensity may vary	≤2 times a month	■ FEV_1 or PEF ≥80% predicted ■ PEF variability <20%

* The presence of one of the features of severity is sufficient to place a patient in that category. An individual should be assigned to the most severe grade in which any feature occurs. The characteristics noted in this figure are general and may overlap because asthma is highly variable. Furthermore, an individual's classification may change over time.

** Patients at any level of severity can have mild, moderate, or severe exacerbations. Some patients with intermittent asthma experience severe and life-threatening exacerbations separated by long periods of normal lung function and no symptoms.

emergency visits or hospitalizations for asthma, and using medication to control asthma with as few side effects as possible. The family needs to be involved in the care of both children as well as adults and an open dialogue between the physician and the patient is essential for successful asthma treatment.

Although it may seem difficult to achieve medication adjustment and asthma education in a 30-minute office visit, the daily diary can serve as a remarkable time-saving tool. A quick review will allow the patient (and/or parent) to highlight difficulties and help the physician focus on the important clinical changes required in management. In addition, the more active a role the patient (and/or parent) plays in management of the disease, the greater likelihood of compliance with medication and avoidance of triggers. Well-placed reminders in the office such as posters illustrating proper technique for metered dose inhaler administration or the technique for peak flow measures can reinforce the message while the patient is waiting in the exam room. In general, the more education during those first visits, the greater the likelihood of disease control long-term.

Asthma is increasing in prevalence throughout the country. Unfortunately, just when more physician-patient interaction is necessary to treat this disease, less time is available. For asthmatics to get the care they need, more efficient assessment, management and education need to occur in the office.

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FIGURE 3 Stepwise Approach for Managing Asthma in Adults and Children Older Than 5 Years of Age

	Treatment		**Preferred treatments are in bold print.**
	Long-Term Control	**Quick Relief**	**Education**
STEP 4 **Severe Persistent**	Daily medications: ■ **Anti-inflammatory: inhaled corticosteroid (high dose)** AND ■ Long-acting bronchodilator: either **long-acting inhaled beta$_2$-agonist,** sustained-release theophylline, or long-acting beta$_2$-agonist tablets AND ■ Corticosteroid tablets or syrup long term (2 mg/kg/day, generally do not exceed 60 mg per day).	■ Short-acting bronchodilator: **inhaled beta$_2$-agonists** as needed for symptoms. ■ Intensity of treatment will depend on severity of exacerbation; see component 3-Managing Exacerbations. ■ Use of short-acting inhaled beta$_2$-agonists on a daily basis, or increasing use, indicates the need for additional long-term-control therapy.	Steps 2 and 3 actions plus: ■ Refer to individual education/counseling
STEP 3 **Moderate Persistent**	Daily medication: ■ Either **Anti-inflammatory: inhaled corticosteroid (medium dose)** OR **Inhaled corticosteroid (low-medium dose)** and add a long-acting bronchodilator, especially for nighttime symptoms: either **long-acting inhaled beta$_2$-agonist,** sustained-release theophylline, or long-acting beta$_2$-agonist tablets. ■ If needed Anti-inflammatory: **inhaled corticosteroids (medium-high dose)** AND **Long-acting bronchodilator,** especially for nighttime symptoms; either **long-acting inhaled beta$_2$-agonist,** sustained-release theophylline, or long-acting beta$_2$-agonist tablets.	■ Short-acting bronchodilator: **inhaled beta$_2$-agonists** as needed for symptoms. ■ Intensity of treatment will depend on severity of exacerbation; see component 3-Managing Exacerbations. ■ Use of short-acting inhaled beta$_2$-agonists on a daily basis, or increasing use, indicates the need for additional long-term-control therapy.	Step 1 actions plus: ■ Teach self-monitoring ■ Refer to group education if available ■ Review and update self-management plan
STEP 2 **Mild Persistent**	One daily medication: ■ **Anti-inflammatory:** either **inhaled corticosteroid** (low doses) or **cromolyn or nedocromil** (children usually begin with a trial of cromolyn or nedocromil). ■ Sustained-release theophylline to serum concentration of 5-15 mcg/mL is an alternative, but not preferred, therapy. Zafirlukast or zileuton may also be considered for patients ≥12 years of age, although their position in therapy is not fully established.	■ Short-acting bronchodilator: **inhaled beta$_2$-agonists** as needed for symptoms. ■ Intensity of treatment will depend on severity of exacerbation; see component 3-Managing Exacerbations. ■ Use of short-acting inhaled beta$_2$-agonists on a daily basis, or increasing use, indicates the need for additional long-term-control therapy.	
STEP 1 **Mild Intermittent**	■ No daily medication needed.	■ Short-acting bronchodilator: **inhaled beta$_2$-agonists** as needed for symptoms. ■ Intensity of treatment will depend on severity of exacerbation; see component 3-Managing Exacerbations. ■ Use of short-acting inhaled beta$_2$-agonists more than 2 times a week may indicate the need to initiate long-term-control therapy.	■ Teach basic facts about asthma ■ Teach inhaler/spacer/holding chamber technique ■ Discuss roles of medications ■ Develop self-management plan ■ Develop action plan for when and how to take rescue actions, especially for patients with a history of severe exacerbations ■ Discuss appropriate environmental control measures to avoid exposure to known allergens and irritants (See component 4.)

↓ **Step down**
Review treatment every 1 to 6 months; a gradual stepwise reduction in treatment may be possible.

↑ **Step up**
If control is not maintained, consider step up. First, review patient medication technique, adherence, and environmental control (avoidance of allergens or other factors that contribute to asthma severity).

NOTE:

- **The stepwise approach presents general guidelines to assist clinical decisionmaking; it is not intended to be a specific prescription. Asthma is highly variable; clinicians should tailor specific medication plans to the needs and circumstances of individual patients.**
- Gain control as quickly as possible; then decrease treatment to the least medication necessary to maintain control. Gaining control may be accomplished by either starting treatment at the step most appropriate to the initial severity of the condition or starting at a higher level of therapy (e.g., a course of systemic corticosteroids or higher dose of inhaled corticosteroids).
- A rescue course of systemic corticosteroids may be needed at any time and at any step.
- Some patients with intermittent asthma experience severe and life-threatening exacerbations separated by long periods of normal lung function and no symptoms. This may be especially common with exacerbations provoked by respiratory infections. A short course of systemic corticosteroids is recommended.
- At each step, patients should control their environment to avoid or control factors that make their asthma worse (e.g., allergens, irritants); this requires specific diagnosis and education.
- Referral to an asthma specialist for consultation or comanagement is *recommended* if there are difficulties achieving or maintaining control of asthma or if the patient requires step 4 care. Referral may be *considered* if the patient requires step 3 care (see also component 1-Initial Assessment and Diagnosis).

All That Wheezes Isn't Asthma:
Why Primary Care Physicians Don't Use the Guidelines for the Diagnosis and Treatment of Asthma

Michael Fine, MD

The challenge of primary care diagnosis and treatment is to turn a patient's report of discomfort, received in their language, into a coherent diagnosis that makes medical sense, and then initiate treatment based on that diagnosis. Primary care physicians see a different universe of complaints from specialty physicians, and only a few of these complaints persist and meet criteria for the diagnosis of disease.

Patients frequently report they have asthma when they may not have had the formal pulmonary testing or office spirometry necessary to establish the diagnosis.

It is easy to confuse a history of asthma with asthma itself. Patients frequently report they have asthma when they may not have had the formal pulmonary testing or office spirometry necessary to establish the diagnosis. (This is particularly true for patients who received the diagnosis as children, a time when the diagnosis is based on symptom presentation and not objective testing.) Patients may have once had asthma but the asthma may have remitted, a situation known to all primary care physicians but not reflected in guidelines, which do not contain recommendations for periodic retesting, as the duration of asthma is not known. Or pediatric patients may have simply received the wrong diagnosis, having presented with recurrent stridor or chronic cough; and years later the patient considers him/herself an asthmatic.

It is also easy to confuse wheezing, dyspnea or chronic cough with asthma, particularly if these complaints occur chronically or recurrently. The differential diagnosis of wheezing presenting to the primary care physicians includes post-infectious wheezing (most common by far); allergic wheezing that does not meet the criteria for asthma, congestive heart failure, gastroesophageal reflux, foreign body obstruction, and cold- and exercise-induced bronchospasm that does not meet the criteria for asthma. The differential diagnosis for dyspnea presenting to the primary care physician includes deconditioning, panic disorder, cardiac disease including active ischemic heart disease, restrictive lung disease, pneumonia and other interstitial diseases of the lung, intra-thoracic neoplasm and sleep apnea. The differential diagnosis for chronic cough includes chronic sinusitis, chronic tracheitis, gastrophageal reflux, congestive heart failure, intra thoracic neoplasm, neoplasm of the head neck or throat, allergic post nasal drip, other interstitial lung disease, and sleep apnea.

In order to make use of these guidelines, then, primary care physicians will need to approach the diagnosis of asthma with new rigor, recognizing that the guidelines are useful only when the diagnostic criteria are fulfilled. It may take many office visits, many diagnostic modalities, and many therapeutic trials to establish the diagnosis of asthma with certainty. These Guidelines are to be used only when the diagnosis is firmly established. It must also be recognized that there are no guidelines for the treatment of non-asthma wheezing, a condition whose effective treatment, at present, depends on the knowledge, skill and art of the treating primary care physician.

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A Conversation: Managing Asthma in Children, Mary Ann Passero, MD, and David Chronley, MD

compiled by Mary Ann Passero, MD

Dr. Chronley:

For several years the asthma experts have re-emphasized the importance of treating airway inflammation as well as bronchospasm in persistent asthma.[1] Apparently inflammation occurs not only during acute exacerbations of asthma but is also present in the airways of patients with even mild daily symptoms.[2]

Dr. Passero:

Yes. It was recently reported that adults who identify themselves as having asthma have substantially greater declines in FEV_1 over time than those who do not.[3] Data from the Childhood Asthma Management Program show that lung function in children also declines faster than normal and the decline is directly related to the duration of the child's asthma.[4] Even children under 4 years of age with a history of persistent wheezing for as little as 2 years may have lost pulmonary function which cannot be completely corrected.[5]

Dr. Chronley:

"Airway remodelling" denotes the loss of airway caliber in asthma secondary to chronic inflammation.[6] Even though airway remodeling may occur very early in the course of the disease, not all asthma patients develop irreversible airway obstruction. It is not even clear that aggressive therapy with anti-inflammatory agents can completely prevent airway remodeling.

Dr. Passero:

All you say is true, but those patients with more severe persistent symptoms who are atopic and especially those patients who have adult relatives with persistent asthma are at greater risk. Several studies have shown that the regular use of inhaled steroids for persistent wheezing will decrease the frequency and severity of asthma exacerbations, reduce the need for emergency room visits, hospitalizations and systemic steroids, and also improve the quality of life for adults and children with persistent asthma.[5-7] Other anti-inflammatory agents such as cromolyn, nedocromil and the leukotriene receptor antagonists are available for children or adolescents but may not always be as effective as inhaled steroids in controlling airway inflammation in some patients.

Abbreviations Used:

BDP	beclomethasone diproprionate
FDA	Food and Drug Administration

Dr. Chronley:

We know that prolonged use of *systemic* steroids is associated with adrenal suppression, cataracts, glaucoma, osteoporosis, growth retardation, immunosuppression and glucose intolerance. We have been told that the relatively small amounts of systemic steroids absorbed while *inhaled* make them much safer for long term use. But now almost 30 years after inhaled steroids were introduced for asthma treatment, there have been several records of systemic side effects related to long term use. Most of the more serious problems are related to prolonged use of inhaled steroids in adults. For us pediatricians, the most disturbing news is that significant growth suppression can be measured in some children after as little as 3 months of use.[7-9]

Dr. Passero:

Although in short term prospective studies the use of inhaled steroids has shown measurable reduction in the growth rates of prepubertal children, long term retrospective studies show that asthmatic children receiving inhaled steroids eventually achieve normal predicted adult height. However, in the longer retrospective studies it is not clear that the inhaled steroids were given consistently in the same dose as in the shorter studies.[10] The data on growth suppression in children is compelling enough for the FDA to put labels on inhaled steroids warning parents and patients on their potential for growth suppression.

Dr. Chronley:

In sufficient doses all currently available inhaled steroid preparations have been shown to cause systemic effects.[9] Some of the systemic effects of beclomethasone diproprionate (BDP) and triamcinolone are the result of oral and gastrointenstinal absorption, which can be reduced to some extent by using spacers and teaching patients to rinse their mouths after use.

Newer inhaled steroid preparations such as flunisolide, budesonide, and fluticasone have "first hepatic pass metabolism," so that the oral and gastrointestinally absorbed drug has minimal systemic effect. However, all the steroid that gets past the bronchi and into the alveoli is systemically absorbed.[9]

Dr. Passero:

New data suggest that each of the available inhaled steroids coupled with an appropriately tested delivery device has a "safe" low dose even for children as young as 4 years of age.[10-13] Although there may be some variations between patients, the data suggest that doses of less than 400 micrograms per day of BDP and budesonide and less than 200 micrograms per day of fluticasone do not cause growth retardation.[10] There is little data to help us with the long term use of inhaled steroids in children under 4 years of age. In addition, concomitant use of nasal steroids may reduce the "safe" dose. We also know that for many young asthma patients these "safe" doses will not be sufficient to consistently control their asthma.

Those patients with more severe bronchospasm and airway inflammation tend to deposit more of the drug in the larger airways where it is useful and less in the alveoli where it leads to systemic effects. On the other hand, as the disease improves, more inhaled steroid is liable to reach the alveoli and be systemically absorbed. Using inhaled steroid primarily in the morning may also reduce side effects.[10]

> *Several studies have shown that the regular use of inhaled steroids for persistent wheezing will decrease the frequency and severity of asthma exacerbations, reduce the need for emergency room visits, hospitalizations and systemic steroid, and also improve the quality of life for adults and children with persistent asthma.*

Dr. Chronley:

For selected patients with mild persistent asthma who are willing to use prophylactic inhalers frequently, cromolyn or nedocromil can be effective and safe. In moderate or severe persistent asthma the addition of these mast cell stabiliziers may reduce the need for higher dose inhaled steroids. Similarly, leukotriene receptor antagonists which have become available in the past 2 years may be effective in selected mild persistent asthma and in combination with inhaled steroids in moderate and severe asthma. Montileukast, a leukotriene receptor antagonist, has been approved for children as young as six years of age; Zafirleukast, for children 12 years of age and above. Leukotriene receptor antagonists appear to be relatively free of severe short term side effects. There are not yet any long term follow up studies addressing safety or efficacy. Since these are oral drugs and need to be taken only once (montileukast) or twice (zafirleukast) a day, compliance is not usually a problem.[14]

Salmeterol is a long-active beta agonist, which has been used as an inhaler twice a day to control night time asthma as well as providing more consistent bronchodilator during the day. Together with an inhaled steroid it has been shown to reduce the need for high dose inhaled steroid to achieve asthma control. Salmeterol in a disc inhaler has become available for children as young as 4 years of age.[15-17]

Long-acting theophylline preparations may also be used in selected patients to achieve asthma control along with low-dose inhaled steroids. They have a narrow therapeutic window and require frequent serum level monitoring. Even in the desired low therapeutic range they may cause attention problems, gastrointestinal upset and headache. Theophylline also has important drug interactions with erythromycin, antifungal agents, zafirleukast, etc.

> *The data on growth suppression in children is compelling enough for the FDA to put labels on inhlaed steroids warning parents and patients ...*

Dr. Passero:

Careful monitoring of the child's symptoms and response to therapy is key to avoiding either under- or over-medication.

Although pulmonary function testing and monitoring of peak flow is useful in children as well as adults, there is no reliable convenient objective measurement of pulmonary function in infants and very young children. Parents both underreport cough and shortness of breath in infants and young children and overreport wheezey and stridorous noises that may be of upper airway origin. The physician must not only assess the child's respiratory status and response to medication using physical signs and symptoms in the office, but must also teach the child's parents which signs and symptoms to watch for and which can be safely ignored.

The older child can be taught to recognize his or her own signs and symptoms and report them appropriately. Peak flow meters can be helpful, but peak flow is quite effort-dependent. Clinical signs and symptoms must be assessed together with peak flow and frequency of need for short-acting bronchodilators to make correct therapeutic adjustments.

Individual and group asthma care classes can reinforce teaching begun in the hospital or office setting. It is not enough to institute home monitoring. The patient and parents must have a written plan to respond to a change in symptoms, signs and/or peak flow reading and a "fail-safe" plan if the change in regimen is not effective. Written plans for the delivery of asthma medications at school, day care, camp, etc., must also be made. Given our concern over the long term use of inhaled steroids, there must be a "step down" as well as a "step up" plan. Communication between the patient, his/her parents and the health care provider is crucial.

For those patients on chronic inhaled steroids, monitoring in the office should consist of height and weight with appropriate growth curves. Peak flow measurements should be obtained at every visit. Timed expiratory flow (FEV_1) is a better measure than peak flow to assess progression of airway disease. Spirometry (before and after inhaled bronchodilator) should be done when the child is old enough to cooperate and periodically thereafter so long as the child continues to require asthma medication. Patients requiring higher dose inhaled steroids will need more frequent spirometry as well as other monitoring. Patients using minimal "safe" doses may require less frequent visits.

During each visit the child's medications, symptoms and response to medication should be re-evaluated and his treatment plan adjusted. Some patients may require more frequent monitoring during certain seasons than in others. Not only medications, but spacers and monitoring plans must be adjusted as the patient grows older and his needs and/or symptoms change. In short, every patient requires his own individual plan.

Dr. Chronley:

We have been talking about medication, but many environmental factors contribute to airway inflammation. Tobacco smoke should always be eliminated from the child's environment. High exposure to viral respiratory infections in a day care or play group setting may play a significant role in the persistence of wheezing in the infant or young child. Changing to a smaller group setting or limiting the number of children under age 3 in the group may help. Scrupulous hand washing, the use of disposable cups or eating utensils and frequent washing of shared toys may also help to reduce the frequency and severity of respiratory infections in day care settings.

Particularly in children, asthma is seen more frequently in atopic patients. Reducing exposure to known allergens (animal danders, dust mites, cockroaches, molds and pollens) can often be done at relatively low cost and without adding medication.

Allergen-proof encasings for mattresses, pillow and boxsprings, eliminating pets, washing bedclothes and plush animals frequently, eliminating "dust collectors," reducing carpeting and upholstery in bedroom and play areas, avoiding strong-smelling perfumes and housing cleaning agents all can be done at relatively little cost.

In selected cases more extensive improvements in ventilation, air-conditioning, humidity reduction or air filtration may be necessary.

Dr. Passero:

The allergist may be of help in identifying allergic triggers. Rarely food allergies may play a role. More ammunition in the form of positive skin testing may be necessary to convince a family to remove a pet. If environmental allergens are a significant asthma trigger and cannot be easily avoided, hyposensitization to selected allergens may be effective.

Dr. Chronley:

Adherence to a comprehensive treatment plan with frequent office visits, a complex daily regimen and frequent monitoring requires great effort on the part of patients and their families as well as health care providers.

Today's families, with two working parents and a complex academic and social schedule for each child, seem to be stretching their limits even without the addition of an asthma regimen. Adolescents, particularly, may be expected to medicate and monitor themselves. There seem to be more lost, stolen or destroyed inhalers, spacers and peak flow meters than those in active use.

Exposure to animals at a sleep-over, or exploring a damp basement or dusty attic, may be dangerous to the asthma patients even though acceptable for everyone else.

Just like diabetes patients, asthma patients do not want to be different from their peers and may deny their illness and stop using prophylactic medication.

A disproportionate number of asthma children belong to minorities and live below the poverty level. Social, economic and psychologic stresses also interfere with adherence to an asthma regimen. Education and detailed therapeutic and monitoring plans may still not prevent a child from being lost to follow-up and reappearing only when in the midst of a severe asthma attack.

> *New data suggest that each of the available inhaled steroids coupled with an appropriately tested delivery device has a "safe" low dose even for children as young as 4 years of age.*

Dr. Passero:

It is precisely these patients who cannot adhere to asthma treatment guidelines who are our greatest challenge. Many of the poorest will at least qualify for medical assistance or RIte Care. Asthma affects over 5 million U.S. children. It is the most common cause for pediatric hospital admissions. It accounts for more lost school days than any other chronic childhood disease.[18] We may hope to offer safer more effective and less complex asthma regimens to children in the future, but for the moment we must address today's asthma with today's tools.

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Leukotriene Modifiers – A New Treatment Option for Asthma

Cynthia A. Wedekind, PharmD

Asthma is a chronic inflammatory condition affecting 14 million to 15 million people in the United States.[1] Despite both improved understanding of the disease and better medication choices, the morbidity and mortality rates continue to rise. In 1997, the National Institutes of Health issued new guidelines for treatment which focus on the prevention of acute episodes by controlling the underlying chronic inflammatory disease.[1] The guidelines acknowledge a role for leukotriene modifiers in the treatment of mild to moderate asthma. However, it remains unclear which patients will benefit most and when to introduce these medications into the treatment strategy. Currently three such agents have been approved for use in the US: zafirlukast, montelukast, and zileuton. Several other agents are under investigation.

Leukotriene modifiers are the first new class of medications to treat asthma in over 20 years. They were developed as an oral alternative to corticosteroids to treat the chronic inflammation associated with asthma. Leukotrienes (LTs) are inflammatory mediators which cause airway obstruction and hyperresponsiveness. Their synthesis begins with the breakdown of membrane phospholipids, resulting in increased vascular permeability and mucus production, and causing a powerful constriction of bronchial smooth muscle.[2] Arachidonic acid (AA) is the key substrate of LT generation. Following its release from membrane phospholipids by hydrolysis, arachidonic acid is metabolized by one of two pathways.[3] The cyclooxygenase pathway results in the production of prostaglandins (PGs) and thromboxane A2 (TXA2). The 5-lipoxygenase (5-LO) pathway produces LTs. Previously identified as "Slow-Reacting Substances of Anaphylaxis," LTs are known to consist of several separate compounds. The dihydroxy LT, LTB4, acts directly at the LTB4 receptor to cause neutrophil chemotaxis, aggregation and activation, leading to the release of superoxide and other mediators.[3] LTB4 has not been found to play a large role in the inflammatory response of asthma. The cysteinyl LTs (LTC4, LTD4, and LTE4) are generated by mast cells and other inflammatory cells. Studies have shown that the exogenous administration of the cysteinyl LTs mediates bronchial smooth muscle contraction, mucus secretion, and vascular leakage.[4] There are two classes of medications acting via LT modulation: the competitive leukotriene receptor antagonists (LTRAs), including zafirlukast and montelukast, and the LT synthesis inhibitors, including zileuton.

Abbreviations Used:

LT	leukotriene
AA	arachidonic acid
PG	prostaglandin
TXA2	thromboxane A2
5-LO	5-lipoxygenase
LTRA	leukotriene-receptor antagonist
FEV_1	forced expiratory volume in one second
PEFR	peak expiratory flow rate
LFT	liver function test
FLAP	5-LO activating protein

Table 1. Summary

	Zafirlukast	Montelukast	Zileuton
Indication	Mild to moderate asthma in patients > 12 years	Mild to moderate asthma in adults and children ≥ 6 years	Mild to moderate asthma in patients > 12 years
Mechanism of Action	-LT receptor antogonist -Inhibits LTD4	-LT receptor antogonist -Inhibits LTD4	-LT synthesis inhibitor -Inhibits 5-LO
Dose	20 mg twice daily on an empty stomach	≥ 15 years: 10 mg daily 6-14 years; 5 mg daily	-600 mg 4 time daily - Take with meals and at bedtime
Adverse Effects	Headache Cough Rhinitis	Headache Cough Upper respiratory infections	Elevated ALT Headache Abdominal pain Myalgias Dyspepsia Nausea
Drug Interactions	Aspirin Erythromycin Terfenadine Theophylline Warfarin	No significant interactions noted	Propreanolol Terfenadine Theophylline Warfarin
Availability	20 mg tablets	5 mg chewtabs 10 mg tablets	600 mg tablets
AWP* 30 day supply	$59.77	$69.50 (5 or 10 mg dose)	$82.33

*AWP is the "average wholesale price" to pharmacies taken from the *Red Book*, Cardinale V, ed., Montvale, NJ, Medical Economics, 1999.

ZAFIRLUKAST (ACCOLATE®)

Zafirlukast, the first leukotriene receptor antagonist approved for use in the US, is indicated for the long-term management of asthma in patients 12 years of age and older. In three 13-week, randomized, double-blind, placebo-controlled trials enrolling over 1300 patients with mild to moderate asthma, zafirlukast demonstrated a statistically significant reduction in daytime symptoms and nighttime awakenings, beta$_2$-agonist use, and improvement in forced expiratory volume in one second (FEV_1) and peak expiratory flow rate (PEFR).[5] Adverse effects with zafirlukast have been minimal, with the largest complaints being headache, rhinitis and cough. Recently, there have been reports linking the use of zafirlukast to Churg-Strauss Syndrome, a rare eosinophilic vasculitis.[6] Only sporadic cases have been reported to date, but this clearly indicates an area where continued research is needed. Zafirlukast undergoes extensive metabolism in the liver via the cytochrome P-450 system, and drug interactions are common (Table 1). The bioavailability of zafirlukast is decreased when administered with food, so it must be taken on an empty stomach.

All three leukotriene modifiers have shown promise in reducing symptoms associated with chronic asthma, offering an alternative to patients currently maintained on inhaled corticosteroids.

MONTELUKAST (SINGULAIR®)

Montelukast is a selective antagonist of the LTD4 receptor. It has been studied and approved for use in both adults and children greater than 6 years of age. Benefits associated with montelukast include once-daily administration and a relatively safe side effect profile. In a 12-week, multicenter, randomized, double-blind, placebo-controlled trial, montelukast demonstrated significant improvement in asthma control.[7] A total of 681 subjects were evaluated in the study, 408 in the treatment group and 273 in the placebo group. Montelukast, 10 mg daily, demonstrated statistically significant improvement of airway obstruction, documented by an increase in FEV_1 of 13.1% (placebo, 4.2%), morning PEFR of 24 L/min (placebo, 4.6 L/min), and evening PEFR of 15.9 L/min (placebo, 4.2 L/min). Patient-reported endpoints, including daytime asthma symptoms, as-needed beta$_2$-agonist use, and nocturnal awakening were all significantly improved in the treatment group. Side effect profiles were similar between the two groups, with the most commonly reported side effects in the treatment group being headache and upper respiratory infections.[7] Montelukast has also been studied in children ages 6 – 14 years. In a two month study of 336 children with chronic asthma, a daily dose of 5 mg of montelukast increased FEV_1 by 8.23% (placebo, 3.58%).[8] Secondary outcomes including daily as-needed beta$_2$-agonist use and quality of life measures were also more favorable in the treatment group. In summary, the studies support the use of montelukast in the management of chronic asthma.

ZILEUTON (ZYFLO®)

Zileuton is a potent selective inhibitor of 5-LO. Unlike the LTRAs, zafirlukast and montelukast, zileuton inhibits the production of both LTB4 and the cysteinyl LTs. The long-term effects of zileuton were evaluated in a 13-week, randomized, double-blind, parallel design study.[9] A total of 401 patients were enrolled in three subject groups: 600 mg zileuton 4 times daily (n=132), 400 mg zileuton 4 times daily (n=134), and placebo 4 times daily (n=135). The 600 mg zileuton group demonstrated statistically significant improvements in FEV_1, asthma exacerbations requiring corticosteroid treatment, and quality-of-life issues. However, elevated liver function tests (LFTs), defined as greater than 3 times normal, were noted in both treatment groups when compared with placebo. All elevations reversed upon discontinuation of the medication.[9] The incidence of elevated LFTs has prompted a warning against the use of zileuton in patients with a history of hepatic dysfunction. All patients receiving zileuton should have LFTs measured prior to initiation of treatment, monthly for the first three months, and periodically thereafter. If an elevation of LFTs (greater than 3 times normal) is noted, the medication should be discontinued. Zileuton is extensively metabolized in the liver and significant drug interactions can occur (Table 1). Zileuton has a half-life of approximately 2 hours. For this reason, it requires a four times daily dosing regimen, which may be difficult for noncompliant patients.

CONCLUSION

All three leukotriene modifiers have shown promise in reducing symptoms associated with chronic asthma. The simple dosing regimens of zafirlukast and montelukast may be advantageous in noncompliant patients. Zileuton may not offer this compliance benefit as its short half-life requires frequent dosing. Zileuton also carries the risk of elevated LFTs, the monitoring of which will add to the overall cost of the medication. The efficacy of these medications in clinical practice remains to be seen.

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Managing Asthma in Rhode Island Schools

Ann Kelsey Thacher, MS, and Rosemary Reilly-Chammat, MPA

INTRODUCTION - OVERVIEW OF PROBLEM

Asthma is increasingly diagnosed in Rhode Island school-aged children. Schools cannot ignore this disease if they want to succeed in their mission, and physicians cannot ignore schools if they want their patients to succeed in managing their asthma. Effective asthma management requires collaboration among student and families, physicians, and school staff. (Table 1)

The National Heart, Lung and Blood Institute (NHLBI), Asthma Education and Prevention Program, School Asthma Education Subcommittee identified seven areas for asthma management in schools: school nursing services; (and health services staff in schools); medication administration; physical education opportunities; emergency plans; smoke free schools; other indoor air quality issues; education for students and staff.

First and foremost, the student's physician must develop an individualized asthma management plan, which enables the child to participate in school activities to the maximum extent. Before leaving the office, the patient (parent) should have this plan in hand. [See Charles Sherman, "Management of Asthma in the Office," this issue.] The implementation of this plan is accomplished through a partnership among the provider, the family, the school nurse and other school personnel, as well as the student. That plan should include: student information; asthma triggers, school environmental control measures, premedication, and/or dietary restrictions; peak flow monitoring schedule; daily medication plan; any special conditions; emergency asthma plan including emergency asthma medication; recommendations on self-administration of medication

The success of any asthma management plan rests on the ability of the child to implement it. Younger children will need assistance, but with older children, families, schools and primary care providers should make "self-management" the goal.

HEALTH SERVICES STAFF IN SCHOOLS

The health professionals with overall and day-to-day responsibility for health services provided in schools should work closely with students' primary care providers. All school districts must have a school physician, licensed in Rhode Island and under contract to the district (RIGL 16-21-9). This physician serves as a consultant to develop school health protocols and provide training for school nurses. School nurse teachers (certified by the Department of Elementary and Secondary Education and licensed as registered nurses in Rhode Island (RIGL 16-21-8)) administer the school health program. (In non-public schools, registered nurses licensed in Rhode Island are considered "substantially equivalent" for individual health care, including dispensing medication in schools.) The school nurse should be a daily educator and reinforcer of a child's asthma education plan and should feel empowered to communicate with children's primary care providers as often as necessary. The school nurse can link school and physician.

Abbreviations Used:

COZs	Child Opportunity Zones
MOB	Majority Over Butts
NHLBI	National Heart, Lung and Blood Institute
SBHC	School-Based Health Centers

MEDICATION ADMINISTRATION

General provisions in Rhode Island law require that school health programs must be carried out by certified school nurse teachers. However, current laws and regulations make no provision for medication administration in schools with the exception of RIGL 16-21-22 and RIGL 16-21-22 which permit students to carry and use prescription epinephrine auto-injectors and inhalers while at school or school-sanctioned events. Within these legal constraints, individual districts have had the latitude to develop their own medication policies.

School nurses usually have assumed the responsibility for medications. They administer, or remind children to self-administer, and reinforce instruction on the use of inhalers and peak flow meters. If the nurse is full-time, these tasks are readily accomplished. If the nurse is at the school part-time, alternative plans need to be developed. Students need the opportunity to take needed medications during the school day, and it is clear from current standards of health care practice that no lay personnel, other than a parent, legal guardian, or the student him/herself, should administer medications.

However, there are currently no statewide regulations that specify policies for administration, storage, self-carry and self-administration of non-prescription or prescription medications in schools (with the exception of epinephrine and inhalers, noted above). The development of such policies is complicated by the commitment of many districts to a "drug free schools" policy.

Under discussion is a new section in the Rules and Regulations for School Health Programs on Medication Administration. The proposed regulations include the following:

- A signed authorization from a parent must be obtained.
- All medications must be stored in their original pharmacy and/or manufacturer labeled containers with the

Table 1.
HOW DOCTORS CAN HELP THEIR SCHOOL-AGED ASTHMA PATIENTS

- Develop a written asthma management plan with the patient and his/her family.
- Learn about the school your patient attends:
  - What are the school policies and protocols about medication administration, emergency treatment, etc.?
  - What are the school resources for health education and health services?
  - Who is the school nurse teacher and what does she know about the patient's management needs?
- Share asthma education materials with patients, their families, and their schools.
- Encourage physical activity in and out of school.

name of the student clearly indicated on the container.

- Principals must be informed of any agreements for medication self-carry and administration.
- All medications to be administered by the school nurse must be stored in a secured cabinet.
- A licensed provider's order shall be obtained and verified by the school nurse.
- School districts must make provisions for self-carry and self-administration of medications prescribed for longer than one month.

At the time of writing, these regulations were under development.

PHYSICAL EDUCATION

Physical activity is important to the health of all children, including those with asthma. Childhood obesity is epidemic; inactive children can develop lifelong patterns putting them at risk for a variety of chronic diseases. Full participation in physical education should be encouraged in the "special condition" portion of the asthma management plan. The only routine contraindication to exercise is when activities take place outdoors during times of high air pollution levels. The physician should advise on the use of any medication before physical activity and the steps to take if asthma symptoms occur during physical activity. Both the student and the school nurse should know those recommendations. If the child develops symptoms, the school nurse should refer the child back to the provider since that indicates a need to modify long-term therapy. Curtailment of physical activity is a last resort in the treatment of asthma.

EMERGENCY PLAN

According to the Rules and Regulations for School Health Programs, each school must have written protocols and standing orders for injuries and acute illnesses, including anaphylaxis (developed by the school physician). In-service basic first-aid training must be provided to school personnel who might be involved in managing a medical emergency. Personnel to be trained are identified by the school principal or other designated school authority; at all times, at least one person, in addition to the school nurse, who is trained in the use of an epinephrine auto injector must be on site. Once again, the school district designs the training protocols. The school physician and school nurse are responsible for implementing the protocols and usually providing the training, so they should be contacted with any questions about the emergency procedures in specific schools.

SMOKE FREE SCHOOLS

One of the most common environmental irritants for asthmatics is tobacco smoke. Since 1992, Rhode Island has banned smoke in school settings (RIGL 23-20.9). The law covers all public and private elementary and secondary schools during and after school hours. It includes classroom buildings, playgrounds, administration buildings, athletic facilities, locker rooms, buses and other school vehicles, and other outside areas within twenty-five feet of any school building. (Teachers cannot have a separate "smoking lounge.")

This law has enabled advocates to demand the enforcement of smoke free schools. By September 1993, all Rhode Island schools were required to have "enforcement" policies. Often those policies merely restate the law. Especially in middle and high schools, there are frequent informal complaints, particularly of smoke-filled student restrooms. Some schools have attempted to supervise restrooms, some use smoke alarms, others have locked them (not a recommended practice); still others have ignored them.

Enforcement of the smoke free school act is variable. The enforcement provision in the law specifies that written confidential complaints citing school violations be directed to the Rhode Island Department of Health for investigation. The Department then serves written notice to the governing body of the school requiring corrective action within ten days. Second complaints for the same or continued violations in the same school result in a referral to the Rhode Island Attorney General, who may assess and recover a civil penalty. Since 1993, there has been only a handful of complaints referred to the Department; all were resolved.

Changing the mindset of students is crucial: it is far easier to make schools truly smoke-free if students themselves want them smoke-free. Chariho High School is an example of student "no-smoking" activism. In the Majority Over Butts (MOB) campaign, students conducted an initiative to enforce the state Smoke Free Schools law. Tired of smoke in their lavatories, students organized a petition drive garnering 770 signatures from other students, and held a demonstration outside the school which increased the willingness of reluctant school administrators to work with students to strengthen the school smoking policy. (One student designed a t-shirt distributed to demonstrators.) The students were supported and guided in their efforts by the school athletic director, who suggested action in response to their complaints about the smoking. The group has produced a video that describes how to implement a MOB project.

INDOOR AIR QUALITY

Aside from tobacco smoke, other common asthma triggers include house dust mites and indoor fungi (molds), and chemical odors from products used for cleaning, in science laboratories or in art classes . [See Robert Vanderslice and Lynn Bibeault, "Asthma and the Environment," this issue.] A 1995 Government Accounting Office report, *School Facilities: Profiles of School Conditions by State*, reported that 30% of Rhode Island schools had unsatisfactory indoor air quality.

Schools, as employers, are governed by OSHA standards, which are enforced by RI Department of Labor and Training. While important for occupational safety, the OSHA regulations address some, but not all of the indoor air quality triggers. The Environmental Health Division of the RI Department of Health can provide technical assistance; school facility managers and local building inspectors also can help.

One provision in the 1997 statute, Article 31 (RIGL 16-7.1), requires schools to have a five-year capital outlay plan (the Asset Protection Plan). The Healthy Schools! Healthy Kids! initiative of the Rhode Island Departments of Health and Education has worked closely with school district facilities' managers to help schools address environmental problems.

Although this has not resolved all indoor air quality problems, long range planning of capital replacement and maintenance is a first step.

Additionally, the Federal Environmental Protection Agency's "Tools For Schools" helps schools develop an indoor air quality management plan. [See Robert Vanderslice and Lynn Bibeault, "Asthma in the Environment," this issue.]

EDUCATION FOR STUDENTS AND STAFF

Primary educators in the schools include school nurse teachers and health educators, and, at the elementary level, classroom teachers. Often teachers are eager to work with community organizations to enhance the health information delivered in schools, and there is a wealth of information on asthma for educators.

Rhode Island's Health Education Framework provides many opportunities to infuse age-appropriate information about asthma to students. Students can learn how to support classmates with asthma, encourage classmates to participate in physical activities and follow their asthma management plan. The Department of Education supports the standards with its Health Education Resource Center. Listings of resources are available on-line. [See Table, "What's Happening in the Community: A Compendium," this issue.]

WAYS TO FILL GAPS

School-Based Health Centers (SBHC) located in five Rhode Island schools (one in Central Falls, Pawtucket, Providence, two in Woonsocket) are also allies in asthma management. The SBHCs have, on-site, medical and behavioral health staff.

Child Opportunity Zones (COZs), located in 19 districts, serve primarily elementary schools. Also with on-site staff, COZs link health and social services to schools and are a vehicle to integrate community resources into schools. (See Table 2)

CONCLUDING RECOMMENDATIONS

Physicians should contact the nurses in the schools which their patients attend.

Physicians can also work with the schools to advocate adherence to policies and laws that impact all children including those with asthma including RIGL 16-7.1, 16-21-22, 23-20.9 and the Rules and Regulations for School Health Programs.

Table 2. Child Opportunity Zone (COZ) Family Center Coordinators

City	Contact	Phone
Bristol-Warren	Joan Ricci	247-3730
Central Falls	Mario Papino	727-7700,x15
Coventry	Sue Conde	822-9400
Cranston	Jeanne Rheaume	943-3029
Cumberland	Rosemarie Crozier	726-2030
East Providence	Grace Osediacz	435-7857
Middletown	Katie Jones	849-2122
Newport	Christine Arouth	845-8579
North Kingstown	Donna Thompson	541-6340
Pawtucket	Mary Parella	729-6293
Providence		
D'Abate	Kathy Hackett	456-1710
Gilbert Stuart	Ramona Rodriguez Mejia	831-5845
Fogarty	Peggy Byrnes	421-0956
Camden	William Bentley	455-3895
Sackett	Aleda Spaulding	461-7940
Woonsocket	Terry Curtin	766-3384
Westerly	Sally Mitchell	596-0315,x218

A comprehensive approach to asthma management, in partnership with school personnel, patients and their families, can ensure that children will be full participants in school. The benefits include reduced absenteeism due to asthma attacks and/or emergency visits; increased participation in physical activity, particularly in physical education classes; increased adherence to physician treatment orders; and adoption of lifelong self care skills.

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What's Happening in the Community: A Compendium

Betina Ragless

A collection of asthma-oriented community resources exists to help patients and families.

American Lung Association of RI (401) 421-6487 or 1-800-LUNG-USA

- General literature on asthma in children and adults, treatment and allergy avoidance.
- Educational seminars for adults and parents of children with asthma held around the state throughout the year.
- Asthma Support Group - for adults and parents of children with asthma.
  The group meets the first Wednesday of the month, October through December and March through June at Evergreen House in East Providence from 7-9 pm.
- School Based Programs
  - A is for Asthma - for children 3-5 years of age and child-care professionals. This educational program, in English and Spanish, features Sesame Street characters Elmo, Rosita, Luis and new Muppet Dani, who has asthma. Caregivers and children learn about asthma as the Sesame Street Muppets sing, dance and talk about Dani's needs. Youngsters will also learn how to help when someone is having trouble breathing.
  - Open Airways for Schools - this program, tested and refined in elementary schools across the country, is a package of easy-to-use teaching materials including a curriculum, instructors guide, poster flip chart, and reproducible handouts for both children and parents in English and Spanish. The program consists of six interactive lessons for children ages 8-11. The package, designed to serve an entire elementary school, can be used over and over.
- Indoor Air Quality Tools for Schools - Easy-to-use kit from the Environmental Protection Agency shows schools how to carry out a practical plan of action at little or no cost using common sense activities and in-house staff to identify and prevent indoor air quality problems. Compliments Open Airways for Schools. [See Robert Vanderslice and Lynn Bibeault, "Asthma and the Environment," this issue.]

Blue Cross/Blue Shield of RI - BlueChip (401)459-5817 or 1-800-637-3718, x 5817

- Easy Steps for Healthy Living - Asthma! - Small informal classes for children, teenagers and adults with asthma, and parents of children with asthma. Participants must attend both of two sessions. Free to Blue Cross/Blue Shield, BlueChip members.

Harvard Pilgrim Health Care of NE (401)331-4034 ext. 42373

- Pediatric and adult small group and one-on-one educational classes in inhaler use, peak flow meter use ar d controlling environment to reduce or avoid common triggers.

Hasbro Children's Hospital (401) 444-8340 or 444-3092 for information

- Draw A Breath Asthma Education- a multi-disciplinary approach for education and advocacy for children with asthma in RI and Southeastern Massachusetts. Weekly programs led by a multi-disciplinary team present lectures and interactive projects on environmental triggers of asthma, and strategies for family and school issues involving asthma. Free spacers and peak flow meters are given families that need them. This year-around program is sponsored by the donations of the Chris and Lisa Van Allsburg family of Providence and Lifespan. No medical insurance is required.
- Draw A Breath Asthma Camp - An overnight camp for children ages 9-13 with moderate to severe asthma. Includes regular camping activities and daily lessons in the care and management of asthma. 24-hour nursing and physician coverage. The Isaac B. Lawton Foundation is the principal sponsor of the camp which is held at the Canonicus Camp and Counseling Center. Many free camperships are available to children with moderate to severe asthma. Lifespan and the ALA donate in-kind support.
- Draw A Breath Childhood Asthma Awareness Week - An annual event held the first week in November offering a series of educational programs for health professionals (physicians, nurses, respiratory therapists and others) and families concerning childhood asthma. Keynote speaker on November 6, 1999: Robert Lemanske, MD, who will be speaking on Viruses and Asthma. Many other events planned.
- Draw A Breath Web Page— a Web site of information and links to important asthma Web resources for RI and Southeastern Massachusetts families with asthma: www.drawabreath.com
- Draw A Breath Community Initiative- a RI community effort funded by the Rhode Island Foundation to provide asthma education to the Providence community at sites near schools throughout the city. It is a collaboration with the organizations "Parents Making A Difference" and the Draw a Breath Asthma Initiative.

Asthma Information on the Internet

(compiled by Rosermary Reilly-Chammat, MPA)

Allergy and Asthma Network (Mothers of Asthmatics, Inc.)
www.aanma.org

American Academy of Allergy, Asthma and Immunology
www.aaaai.org

American Association of Respiratory Care
www.aarc.org

American College of Allergy, Asthma, and Immunology
www.allergy.mcg.edu

American Lung Association
www.lungusa.org

ARA: Asthma Education Resources on the Web
222.arai.com/asthmaed

Asthma, Air Quality in Schools
cnet.unb.ca/cap/partners/chsptf/air_quality

Asthma and Air Quality: A Survey of Schools
www.nfer.ac.uk/summary/asthma

Asthma and Allergy Foundation of America
www.aafa.org

Asthma Information Center
www.mdnet.de/asthma

Asthma and Allergy Information and Research (AAIR)
www.users.globalnet.co.uk/~air

Asthma and School
www.allergyasthma.com/archives/asthma04

Asthma Society
www.asthmasociety.com/manual

Canadian Lung Association
www.lung.ca/asthma/index.html

Daily Lung Newspaper
www.dailylung.com

Food Allergy Network
www.foodallergy.org

JAMA Asthma Information Center
www.ama-assn.org/special/asthma

Journal (asthma) articles
www.mediconsult.com/asthma/journal/content.html

National Asthma Education & Prevention Program, NHLBI
www.nhlbi.nih.gov/nhlbi

National Jewish Medical and Research Center
www.njc.org

National Education Association
www.nea.org/hin/air

New York City Department of Health: Open Airways for Schools
www.ci.nyc.ny.us/html/scah/openair.html

Open Airways for Schools
ehpnet1.niehs.nih.gov/docs/1996/104-5/iti.html

Rhode Island Department of Education, Health Education Resource Center
www.ri.net/RIDE/health/bibliography

Rhode Island Department of Health
www.health.state.ri.us/regs

U.S. Department of Education
www.ed.gov/offices/ocr

U.S. Environmental Portection Agency
www.epa.gov/iaq

University of Iowa
vh.radiology,uiowa.edu/...gingAllergies

University of Texas-Houston
www.uth.tmc.edu/uth_org...rs/uthouston/feb_96/inner

Neighborhood Health Plan of RI (401)459-6000 or 1-800-963-1001

- Pediatric and adult asthma education programs in small groups or one-on-one in the home. Adults and children are taught use of inhalers and peak flow meters, and environmental control of triggers.

United Health Plans of NE (401)737-6900 or 1-800-822-3807

- Pediatric Asthma Care and Education (PACE) Program - An in-home assessment by a respiratory care practitioner to tailor an educational plan for the individual child and family on early warning signs and symptoms, medications, triggers and school care. Can be provided in languages other than English.
- Asthma Education (ACE) Program - In-home assessment of an adult with asthma (and/or COPD) by a respiratory care practitioner to provide education on the respiratory system, exercises, relaxation techniques, use of inhalers, and nutrition. Can be provided in languages other than English.

Available to United Health Plan members only.

St. Michael's/Harvard Pilgrim Health Plan of New England/ Private Practice Physicians

- "Taming Asthma" Program - This free clinic for adults and children who are under/uninsured offers one-on-one and small group asthma instruction on medication use, environmental issues, and patient peer education. Clinic open 1 evening a month. Located at BroadMed, 557 Broad Street, Providence, RI . phone: (401) 273-1888

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CENTER TABLE 1. Sample Questions* for the Diagnosis and Initial Assessment of Asthma

A "yes" answer to any question suggests that an asthma diagnosis is likely.

In the past 12 months,…

- Have you had a sudden severe episode or recurrent episodes of coughing, wheezing (high-pitched whistling sounds when breathing out), or shortness of breath?
- Have you had colds that "go to the chest" or take more than 10 days to get over?
- Have you had coughing, wheezing, or shortness of breath during a particular season or time of the year?
- Have you had coughing, wheezing, or shortness of breath in certain places or when exposed to certain things (e.g., animals, tobacco smoke, perfumes)?
- Have you used any medications that help you breathe better? How often?
- Are your symptoms relieved when the medications are used?

In the past 4 weeks, have you had coughing, wheezing, or shortness of breath…

- At night that has awakened you?
- In the early morning?
- After running, moderate exercise, or other physical activity?

*These questions are examples and do not represent a standardized assessment or diagnostic instrument. The validity and reliability of these questions have not been assessed.

CENTER TABLE 2.

Patient Self-Assessment: Example of Patient Diary

Date	Wheeze	Cough	Activity	Sleep	Quick Relief: Beta$_2$ – Agonist	Cromolyn/Nedocromil	Inhaled Steroids	Other – Inhaled	Oral Steroids	Theophylline	Peak Flow			Comments
											AM	PM	Other Times	

Wheeze	None = 0	Some = 1	Medium = 2	Severe = 3
Cough	None = 0	Occasional = 1	Frequent = 2	Continuous = 3
Activity	Normal = 0	Can run short distance or climb 3 flights of stairs = 1	Can walk only = 2	Missed school or work or stayed indoors = 3
Sleep	Fine = 0	Slept well, slight wheeze or cough = 1	Awake 2-3 times, wheeze or cough = 2	Bad night, awake most of the time = 3

* Adapted with permission from Plaut 1991.

This diary is provided as an example for clinicians.

CENTER TABLE 3.

Sample* Patient Self-Assessment Sheet for Followup Visits

Name:______________________________ Date:____________________

How many days in the past week have you had chest tightness, cough, shortness of breath, or wheezing (whistling in your chest)?	___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7
How many nights in the past week have you had chest tightness, cough, shortness of breath, or wheezing (whistling in your chest)?	___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7
Do you perform peak flow readings at home?	___ yes ___ no
If yes, did you bring your peak flow chart?	___ yes ___ no
How many days in the past week has asthma restricted your physical activity?	___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7
Have you had any asthma attacks since your last visit?	___ yes ___ no
Have you had any unscheduled visits to a doctor, including to the emergency department, since your last visit?	___yes ___no
How many puffs of your short-acting inhaled beta$_2$-agonist (quick-relief medicine) do you use per day?	____________________ Average number of puffs per day
How many of your short-acting inhaled beta$_2$-agonist inhalers did you go through over the past month?	____________________ Number of inhalers in past month

What questions or concerns would you like to discuss with the doctor?

____

____

____

How well controlled is your asthma in your opinion?	___ very well controlled ___ somewhat controlled ___ not well controlled
How satisfied are you with your asthma care?	___ very satisfied ___ somewhat satisfied ___ not satisfied

*These questions are examples and do not represent a standardized assessment instrument. The validity and reliability of these questions have not been assessed.

CENTER TABLE 4.

From: *Facts About Controlling Asthma*, National Asthma Education and Prevention Program, National Heart, Lung, and Blood Institute, NIH Publication No. 97-2339 A Reproducible Handout

ASTHMA MANAGEMENT PROGRAM • ACTION PLAN

PATIENT NAME	D.O.B.	HPHC NUMBER
ADDRESS	MO	PHYSICIAN NAME
	FA	PHYSICIAN PHONE NO.
PHONE (HOME)	PHONE (WORK)	PROVIDER NUMBER

PHYSICIAN ADDRESS

GREEN

GREEN ZONE: Doing Well

- No cough, wheeze, chest tightness, or shortness of breath during the day or night
- Can do usual activities

And, if a peak flow meter is used,
Peak flow: more than ________
(80% of more of my best peak flow)

My best peak flow is: ________

Take These Long-Term-Control Medicines Each Day. Use Spacer with Inhalers (Include an anti-inflammatory)

Medicine	How much to take	When to take it

Before Exercise ☐ ________ ☐ 2 or ☐ 4 puffs 5 to 60 minutes before exercise

YELLOW

YELLOW ZONE: Asthma is Getting Worse

- Cough, wheeze, chest tightness, or shortness of breath, or
- Waking at night due to asthma, or
- Can do some, but not all, usual activities

-OR-

Peak flow: ______ to ______
(50% - 80% of my best peak flow)

FIRST **Add: Quick-Relief Medicine, Use Spacer - and keep taking your GREEN ZONE medicine**

________ ☐ 2 or ☐ 4 puffs, every 20 minutes for up to 1 hour
(short-acting beta$_2$-agonist) ☐ Nebulizer, once

SECOND **If your symptoms (and peak flow, if used) return to GREEN ZONE after 1 hour of above treament:**

☐ Take the quick-relief medicine every 4 hours for 1 to 2 days.

☐ Double the dose of your inhaled steroid for ________ (7-10) days. -OR-

If your symptoms (and peak flow, if used) do not return to GREEN ZONE after 1 hour of above treatment:

☐ Take: ________ ☐ 2 or ☐ 4 puffs or ☐ Nebulizer
(short-acting beta$_2$-agonist)

☐ Add: ________ mg. per day For ______ (3-10) days
(oral steroid)

☐ Call the doctor ☐ before / ☐ within ________ hours after taking the oral steroid.

RED

RED ZONE: Medical Alert!

- Very short of breath, or
- Quick-relief medicines have not helped, or
- Cannot do usual activites, or
- Symptoms are same or get worse after 24 hours in Yellow Zone

-OR-

Peak flow: less than ________
(50% of my best peak flow)

Take this medicine:

☐ ________ ☐ 4 or ☐ 6 puffs or ☐ Nebulizer
(short-acting beta$_2$-agonist)

☐ ________ mg.
(oral steroid)

THEN CALL YOUR DOCTOR NOW. DO NOT DRIVE. Go to the hospital or call for an ambulance if:

- You are still in the Red Zone after 15 minutes and
- You have not reached your doctor.

WORSENING DANGER SIGNS

- Trouble walking and talking due to shortness of breath
- Lips or fingernails are blue

- Take ☐ 4 or ☐ 6 puffs of your quick-relief medicine AND
- **DO NOT DRIVE.** Go to the hospital or **CALL 911 NOW.**

ADDITIONAL INFORMATION	AUTHORIZING PCP SIGNATURE	DATE

Asthma and the Environment:
A Physician's Guide to Resources, Research and Data

Robert Vanderslice, PhD, and Lynn Bibeault, MS

ENVIRONMENTAL CAUSES AND CONTRIBUTING FACTORS

Environmental exposures are both causal factors for the onset of asthma and contributing factors for the development of the disease in predisposed individuals. The onset of asthma is caused by factors that sensitize the airways. Inhaled allergens, such as those from mites, animal fur, fungi and pollen, are the most common sensitizers. Population studies strongly suggest that allergens may cause the onset of asthma by continuously stimulating chronic allergic inflammation of the airways. As an example, in Barcelona, epidemics of asthma exacerbations were traced to days when soybeans were loaded without a filter. This raised awareness that small amounts of airborne allergen can cause major changes in the lungs of sensitized people. In addition, those treated at the hospital were already allergic to the dust, suggesting that sensitization can occur at low atmospheric concentrations.

The most common indoor allergens are house dust mites, animal allergens, cockroach allergen, and fungi. Mites feed on human and animal scales and are often buried deep in carpets, mattresses and soft furnishings. Mite allergen is found in mite bodies, secretions and excretions. Mite allergen is the main source of allergen in dust. One study has correlated exposure to domestic mites in the first year of life to subsequent development of asthma.

Animal allergens are found in saliva, urine, feces, and dander of warm-blooded animals. Cats are potent sensitizers. Other sources of animal allergens include dogs, pet rodents (often kept in children's rooms) and wild mice or rats in some inner-city areas.

Sensitization to cockroach antigen may be more common than sensitization to dust mite antigen in some locations and among some ethnic groups. Molds and yeasts also act as indoor allergens. *Alternaria* has been established as a risk factor for asthma and been associated with the risk of asthma death in the U.S.

Outdoors, pollens and fungi are the most common allergens that cause asthma in susceptible people. Pollen-induced asthma exacerbations seem to be caused by small particles of starch granules that are released from pollens, particularly, after rainfall. *Alternaria* and *Cladosporium* (which are also indoor fungi) are the only fungi that have been established as risk factors for asthma, tending to be seasonal.

Occupational sensitizers probably represent the only firmly documented cause of asthma in adults. Occupational agents causing asthma include animal proteins (e.g., poultry mites in poultry workers), plant proteins (e.g., flour and amylase in bakers), inorganic chemicals (e.g., persulfate in beauty shops, and stainless steel fumes in welding), and organic chemicals (e.g., glutaraldehyde in hospitals).

Contributing factors increase the likelihood of the devleopment of asthma once exposure to a causal factor has occurred. They may increase susceptibility to asthma. Contributing factors from the environment include passive smoking, active smoking in some occupational settings, air pollution and indoor air pollutants.

Passive smoke (also known as environmental tobacco smoke or second-hand smoke) has been linked to increased respiratory symptoms in children as well as an increased risk for asthma and exacerbations of asthma. Parental smoking has been associated with increased respiratory morbidity, particularly during the first two years of life. Risk is increased with maternal smoking. There appears to be an increase in the risk of asthma in children whose parents smoke, but studies have not confirmed this.

Active smoking is unlikely to be a risk factor for the development of asthma in general. However, for some workers exposed to certain sensitizers, smoking may increase the risk of developing occupational asthma.

In heavily polluted cities, outdoor air pollutants (e.g., sulfur dioxide, ozone, and nitrogen oxides) can trigger bronchoconstriction, transiently increase air responsiveness, and enhance allergic responses. In theory, therefore, pollution may contribute to the development of asthma. However, epidemiological studies attmepting to link the rising trend in asthma with outdoor air polluton have been inconclusive. Some believe that it is possible that chronic exposure to pollution may predispose respiratory disease in a more subtle and complicated manner.

Indoor air pollution has been linked to the development of asthma by some data, but more research is needed. The following are among problems related to indoor air pollution: Respirable particles (from cooking or heating with wood, coal, or kerosene) have been associated with nose irritation, respiratory infections and bronchitis, and lung cancer. Nitrogen oxides (from cooking or heating with natural gas, liquid propane, wood, coal, or kerosene) can cause nose irritation, impaired lung function, and increased infections in children. Formaldehyde (from foam, glue, fireboard, pressed board, plywood, particleboard, carpet backing, and fabrics) has been shown to cause difficulty in breathing and asthma symptoms.

ENVIRONMENTAL TRIGGERS

Triggers cause asthma exacerbations by inducing inflammation or provoking acute bronchoconstriction or both. Some triggers (like irritant gases) cannot cause asthma to develop initially, but can exacerbate asthma once it is present. Other triggers (like allergens and occcupational agents) are further exposures to causal factors that have already sensitized the airways of the person with asthma.

It has recently been discovered in incidents such as the one in Barcelona that very small amounts of airborne allergens are able to cause asthma exacerbations and major changes in the lungs of sensitized people. Therefore, once a person is sensitized, indoor or outdoor allergens can cause asthma exacerbations.

Maternal smoking increases the requirements for medication and emergency room visits for children with asthma. Other irritants that may exacerbate asthma include wood smoke, household sprays, volatile organic compunds (such as polishes and cooking oil) and air pollution. Sulfur dioxide can trigger a dose-dependent airflow limitation in patients with asthma, although it has no effect on the airways of normal subjsects up to very high concentrations. Sulfur dioxide may cause airflow limitation at concentrations as low as 1ppm, a level that may encountered in the workplace.

(Source: National Institutes of Health, *Global Initiative for Asthma*, January 1995, NHLBI Publication 95-3659).

INFORMATION

The patient, family, and clinician can learn more about environmental irritants from these sources:

- National Institutes of Health, Global Initiative for Asthma. January 1995; NHLBI Publication 9 5-3659.)
- Greater Boston Physicians for Social Responsibility, Their web site, "Air Pollution and Primary Care Medicine," (www.psr.org/breathe.htm) references the literature relating air pollution to lung disease, including asthma, with details on specific sensitizers (ex. isocyanates), irritants and nonspecific sensitizers (ex. ozone, sulfur dioxide, and nitrogen dioxide), and adjuvants (ex. diesel exhaust, second had smoke) and their impact on asthmatics.
- The Massachusetts Medical Society
  The Society produces a curriculum on environmental asthma and a talk/slide show with annotated references. Contact: Jeff Dickey, MD, Community Health Center of Franklin County, 338 Montague City Road, Turners Falls, MA 01376; (413) 772-3748; fax (413) 772-2940; email: jdickey@massmed.org
- Plant Guides for Identification of Pollen Triggers
  Asthma patients who are sensitive to the pollen of certain plants can identify those plants and the seasons for producing the most pollen.
  - Newcomb's Wildflower Guide has an excellent key system for identifying plants in bloom. Available at the Audubon Society gift shop in Smithfield, Rhode Island.
  - The New England Wild Flower Society, Garden in the Woods. 180 Hemenway Road, Framingham, MA 01701-2699 (508/877-7630) (http://www.newfs.org) library and bookstore stocks fields guides and information about local plants, including bloom times.
- Rhode Island Department of Environmental Management. The RIDEM Office of Air Resources (222-2808) has available literature on air quality, including the health effects of air pollutants.

REGULATORY/MONITORING ACTIVITIES

The government has taken steps to regulate or monitor the levels of certain triggers.

- *Ground-level ozone*
  Ground-level ozone can build to build to irritating levels in the hot summer. The state Department of Environmental Management and the Rhode Island Department of Health jointly issue Ozone Alerts to the press on days when the ozone levels may be high enough to cause respiratory distress, particularly in children, people who work or exercise outdoors, and people with asthma and other lung conditions.
  RIDEM posts a 'Daily Ozone Forecast' on the Office of Air Resources (www.state.ri.us/dem/ozone/ozoneday.htm.)
  The Environmental Protection Agency Ozone Mapping System gives forecasts for all Northeast states (www.epa.gov/region01/oms/.)
  Contact: Lennie Giuliano 222-2808 x 7041

- *Second-hand Smoke*
  Asthmatics can be particularly troubled by environmental tobacco smoke, which may irritate their airways and/or heighten their response to other allergens.

The Rhode Island Department of Health (RIDOH) administers three salient laws:

– Smoking in Public Places (RI General Laws 23-20.6) passed 02/10/99

Smoking is not permitted in any of the following places used by or open to the public: the state house, elevators, indoor movie theaters, libraries, art galleries, museums, concert halls, auditoriums, buses, primary, secondary or post secondary school buildings, colleges and universities (including dormitories), and public hallways in court buildings, hallways of elderly housing complexes, supermarkets, medical offices and public laundries.

A person in charge of a public area listed above must make reasonable efforts to prevent smoking and post "No Smoking" and warning signs conspicuously. The Attorney General may fine a non-compliant facility $50-$500/day.

Eating facilities (excluding night clubs, lounges, dance clubs, and privately sponsored social affairs) with a seating capacity of fifty or more persons must have separate seating for nonsmokers and smokers. Appropriate arrangements must be made to ask patrons their preference prior to being seated. The person in charge of the facility must post signs as follows:

(A) At the entry stating that the establishment is required by law to have a no-smoking section; and

(B) In the smoking sections identifying the area.

– Workplace Smoking Pollution Control Act (RI General Laws 23-20.7)

Each RI employer must maintain a written smoking policy that states that any nonsmoking employee may object to his/her employer about the smoke hazard or discomfort in the workplace. Using existing means of ventilation or separation or partition of the work space:

(1) The employer shall attempt to reach a reasonable accommodation to protect the nonsmoking employees and to ensure a comfortable environment for all employees.

(2) If the employer cannot make such accommodation, he must prohibit smoking in those areas of the workplace where nonsmoking employees may reasonably be expected to be adversely affected by passive smoke.

The employer's smoking policy shall be announced within three weeks of adoption and posted conspicuously.

> *Environmental exposures are both causal factors for the onset of asthma and contributing factors for the development of the disease in predisposed individuals.*

– Smoke-Free Schools (RIGL 23-20.9)

The law bans smoking in schools and at school events. See Ann Kelsey Thacher and Rosemary Reilly-Chammat, "Managing Asthma in the Schools," this issue

COMPLAINT INVESTIGATION

The Office of Compliance and Inspection (222-1360) can respond to complaints about outdoor air quality, like odor and smoke.

Carbon Monoxide and Nitrogen Dioxide in Ice Arenas

In 1992, after skaters in several incidents were overcome by carbon monoxide from gas-powered ice resurfacing equipment and heaters, RI joined Minnesota as the second state to regulate air quality in ice arenas. Since 1992, no carbon monoxide poisonings related to ice arena have been received. However, simply tuning engines to reduce carbon monoxide may actually increase levels of nitrogen dioxide, which can cause airway hyper-reactivity. Given this information, and no additional regulatory requirements, most arena managers have either replaced their gas-powered ice resurfacing equipment or plan to do so, eliminating the most significant source of dangerous fumes in these arenas. At a cost of $75,000 to $100,000 per machine replaced, this represents a serious voluntary commitment to improving air quality, which exceeds regulatory requirements.

RI DOH attributes this success to the collaboration in drafting both the law and regulations and implementing the program. Arena managers were assembled to discuss the carbon monoxide poisoning events and plausible solutions before attempts were made at regulation. These managers directed the crafting of the law and regulations to best suit the real circumstances of their work. In addition, managers were asked what they needed to implement the program and technical assistance was tailored to these needs.

Contact: Robert Vanderslice, Ph.D. 222-4948 x 2103

Indoor Air Quality in schools: Tools for Schools

Asthma is a cause of chronic absenteeism in schools. The American Lung Association estimates that there are an average of two asthmatic children in each U.S. classroom, sometimes higher in urban schools. Unfortunately, New England states are among the worst ten for self-reporting environmental problems in schools (U.S. General Accounting Office Reports on School Facilities.)

An EPA program addresses poor indoor air quality in schools. Three years ago, the Regional Indoor Environment Program introduced EPA's easy-to-use kit "Indoor Air Quality Tools for Schools" at the New England Asthma and Allergy's conference for school nurses. EPA and its local partners have sponsored over 40 workshops and conferences introducing Tools for Schools, distributing over 1000 copies of the kit. EPA is tracking their use.

The Tools for Schools kit (cosponsored by the National PTA, National Education Association, Association of School Business Officials, American Federation of Teachers, Council for American Private Education and the American Lung Association) offers a practical plan to prevent and resolve indoor air problems at little or no cost using in-house staff. The kit includes checklists, a flexible step-by-step guide for coordinating the checklist and sample policies and strategies to get information out to the whole school community. EPA, in conjunction with the popular TV series, This Old House, has produced a short video (filmed at Dedham, MA, high school) about how to operate ventilation systems in schools.

Eugene Benoit and MaryBeth Smuts of EPA Region I (Boston) report success in assisting schools to implement Tools for Schools, particularly when working with the state Committee for Occupational Safety and Health (COSH) groups and when responding to parental concerns regarding problem schools. Several schools and school systems in each New England state are now recruited as pilot programs. Eugene and MaryBeth have focused on the largest school districts in New England, but have assisted in establishing IAQ teams in smaller rural districts as well. Those Rhode Island school districts with pilots are Bristol County and Providence.

In Vermont, a state legislative committee is considering adopting the Tool for Schools approach for every school. Contacts: Eugene Benoit (617) 918-1639; MaryBeth Smuts, Ph.D. (617) 918-1528

RESEARCH EFFORTS

Because asthma is a baffling epidemic linked to the environment, researchers are investigating the linkage. The following are local research initiatives:

- **Brown University Center for Environmental Studies lead/asthma link**

Are the same homes causing both childhood lead poisoning and asthma? The Center for Environmental Studies previously demonstrated that homes of lead poisoned children were more likely to have code violations, environmental violations, and section 8 subsidies. These indicators may provide inexpensive means to identify housing most likely to cause health problems, allowing focused targeting of prevention and remediation.

Kim Mowery has investigated the link between addresses which have been both the home of a child with lead poisoning and a person hospitalized for asthma.
Contact: Kim Mowery at Kimberly_Mowery@brown.edu

- **National Cooperative Inner City Asthma Study**

The National Cooperative Inner City Asthma Study , sponsored by the National Institute of Allergy and Infectious Diseases (NIAID) of the National Institutes of Health, seeks to identify risk factors for severe asthma in children in inner cities, design an intervention program to reduce the risk factors and monitor improvement. Factors being studied are indoor allergens (dust mite, cat, cockroach), tobacco smoke, and indoor pollutants.

Findings from the first two phases of this study are available. In Phase I, which included 1,500 children, a number of factors were found to be associated with asthma severity, including high levels of indoor allergens (especially cockroach allergen); high levels of smoking among family member and caretakers; and exposure to high levels of nitrogen dioxide, a respiratory irritant. In addition, over half of the participants found it difficult to get follow-up care for their asthma.

In Phase 2, more than 1,000 high risk children and their families were assisted by a nurse practitioner in managing the child's condition and instituting environmental controls, such as the removal of cockroach allergens from their homes. Over 90% of enrolled families complied with the intervention, which led to a striking reduction of asthma symptoms, better school attendance, and a 30% decrease in asthma-related hospitalizations and unscheduled physician and emergency visits for asthma. (http://www.niaid.nih.gov/director/art1h.htm)
Contact: Sahi Rafiullah (301) 496-6752

- **Tufts/BU study of indoor environmental factors in Boston Public Housing**

This community-based project initiated by Tufts University School of Medicine and Boston University School of Public Health included the South Boston Community Health Center and the Tenants Task Force at the West Broadway Housing Development. The project consisted of development of a survey instrument, collection and analysis of data, development of follow-up plans, implementation of action items and refinement and dissemination of the model to other public developments in Boston.

In short, tenants and health center staff were trained about the indoor environment, including environmental hazards and health problems, such as asthma. Together, the university researchers and community partners developed, field-tested and used a 142 question survey that covered a range of environmental and safety issues. Fifty surveys were completed; the results revealed that priority concerns were heating problems, moisture from leaks and the biological growth that moisture promotes, child physical safety and smoking (half of respondents reported being smokers). In addition, 26% of adult respondents reported having been diagnosed with asthma by a doctor.

These findings led to a series of follow-up steps. The South Boston Community Health Center has undertaken an expanded training program about the indoor environment for tenants of the West Broadway Housing Development. Boston Medical Center has begun to collect exposure data on biological and chemical factors in the homes of asthmatic tenants. The Boston Housing Authority is replacing the heating system. The university partners are replicating the project at the Franklin Hill Public Housing Authority.

An unpublished manuscript, *Public Health in Public Housing: A Community-Based Survey of Residents*, by H.Patricia Hynes, Doug Brugge, Julie Watts and Jody Lally, describes this project. Contact: Doug Brugge at Dbrugge@aol.com

DATA SOURCES ON ASTHMA IN RI

Although physicians are not required to report asthma to the Health Department, researchers can find data on asthma from the following government-collected sources (with considerations for medical confidentiality and Institutional Review Board approval):

- Deaths

Data on causes of death are maintained at the RI Department of Health, Office of Vital Records. In 1993, there were 15 deaths with asthma as the cause; the youngest was 45 years old. Contact: Roberta Chevoya at 222-2812

- Health Interview Survey

This telephone-based survey includes 6,683 people from 2,580 households, interviewed in 1996. (See Jay S. Buechner, PhD, and William J. Waters, Jr., PhD, "Prevalence of Asthma in Rhode Island," *Medicine & Health/Rhode Island,* October, 1998.) Information was collected on demographics, social and economic characteristics, coverage for health care costs, and general and specific measures of health status. For those reporting asthma, respondents were asked whether they had been diagnosed by a physician. Information on activity limitations was also collected. Addresses are not available to researchers. Data is available by census tract.
Contact: Jana Hesser, PhD, at 222-2550

- Hospital Discharges

Discharges of patients staying in the hospital for three or more days must be reported to the Department of Health. Information is reported by census tract only, without addresses. Contact: Jay Buechner, PhD, at 222-2550

- **Rhode Island Kids Count Factbook**

Rhode Island Kids Count is expanding data collection on asthma. This year there will be a full indicator on asthma based on hospital discharge data reported to the Rhode Island Department of Health. Discharges will be tabulated by city and town for the state. Contact: Ann Marie Harrington at 351-9400

Robert Vanderslice, PhD, is Chief, Office of Environmental Health Risk Assessment, Rhode Island Department of Health.

Lynn Bibeault, MS, is Deputy Chief, Office of Environmental Health Risk Assessment, Rhode Island Department of Health.

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Upcoming Asthma Summit

RHODE ISLAND'S NEWLY-FORMED ASTHMA CARE AND ADVOCACY NETWORK plans to launch a spring 2000 asthma summit, where clinicians, researchers, patients and families can gather to share information. Spearheaded by the American Lung Association of Rhode Island, this summit draws sponsors from the state's health insurers, the State Department of Health, the Medical Society and the Rhode Island Public Health Association. For the Asthma Network, this summit wil be the first of various educational, clinical and research initiatives geared at improving the treatment of asthma in the state.

FOR ADDITIONAL INFORMATION on the summit and/or network, contact Tina Ragless by phone: (401) 421-6487, fax: (401) 331-5266, e-mail: alaofri@aol.com

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The Asthmatic Worker and the Americans with Disabilities Act

Bruce I. Kogan, JD, LLM

The past forty years have witnessed the expansion of federal anti-discrimination laws into areas never envisioned in the early days of the civil rights movement in the 1950s and 60s. The Voting Rights and Civil Rights Acts of the mid-1960s eventually led to enactment of the Americans with Disabilities Act (A.D.A.) in 1990. Physicians play a significant role under this federal disability discrimination law (and its state human rights counterparts) since patients qualify for protection from employment and other discrimination only if they meet the statutory definition of having a "disability." Although nearly 15 million Americans have asthma or related chronic respiratory conditions, they may have difficulty establishing their entitlement to protection under the A.D.A. unless treating physicians understand their role in the disability legal process.

Abbreviations Used:	
ADA	Americans with Disabilities Act

The A.D.A. prohibits covered employers from engaging in discrimination against qualified individuals with a disability in regard to hiring, advancement, discharge, compensation, or other terms and conditions of employment. To be protected by this law, an individual must be both a "qualified individual" and must have a "disability" as these terms are defined under the act. To be a qualified individual with a disability, one must be able to perform the essential functions of the employment position, with or without reasonable accommodations. The term disability means that the individual has a physical or mental impairment that substantially limits one or more major life activities, or has a record of such an impairment, or is regarded by the employer as so impaired.

Regulations promulgated under the A.D.A. help flesh some of this language out in ways that should cover asthma or other chronic respiratory diseases since the regulations define physical impairment to include any physiological disorder or condition affecting the respiratory system. The physical impairment must substantially limit one or more major life activities to amount to a disability, with "major life activities" defined to mean "functions such as caring for oneself, performing manual tasks, walking, seeing, hearing, speaking, breathing, learning, and working [emphasis added]."

Asthmatic workers who are exposed in the workplace to allergens, irritants, or other triggers of respiratory distress may expect that their employers are required by the A.D.A. or by provisions of the earlier Rehabilitation Act to provide them with reasonable accommodations to their disability. Unfortunately, employees who suffer from asthma have not always succeeded in receiving favorable treatment either from their employers or from the courts. In a series of recent federal court cases involving workplace exposure to either cigarette smoke, paint fumes, other chemical vapors, or just poor ventilation, the employees, more often then not, have been unsuccessful. The courts have denied relief for varied reasons, but in some instances the treating or diagnosing physician's report and recommendations as to accommodations might have been more helpful.

An asthmatic power plant employee whose exposure to stack gas, paint fumes, and smoke, had led to chemical bronchitis requested an allergen-free work environment in Cassidy v. Detroit Edison Co., 138 F. 3d 629 (6th Cir., 1998). The employer initially tried to accommodate the employee by transferring her to a different department, allowing her to use a breathing machine, and scheduling maintenance during her absence. These measures were insufficient to remedy her breathing difficulties. She eventually brought suit against the company for failure to reasonably accommodate her under the A.D.A. One of the physicians who examined the claimant reported that her condition remained dire with extremely poor prognosis and recommended that the claimant's work environment be neither hot nor extremely cold and "free of allergens as is reasonably possible." The court denied her request for relief holding that her "proposed accommodation for essentially an allergen-free workplace, which Defendant attempted to locate within the company, was simply too vague to reasonably inform Defendant of a reasonable accommodation, or was otherwise simply unavailable."

Although nearly 15 million Americans have asthma or related chronic respiratory conditions, they may have difficulty establishing their entitlement to protection under the A.D.A. unless treating physicians understand their role in the disability legal process.

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Exposure to poor ventilation or even specific workplace irritants such as chemical fumes or cigarette smoke have also proven to be insufficient bases for relief under federal or state disability discrimination laws where the doctors' reports concluded that the asthmatic employee was disabled from working in particular locations, but not substantially limited in breathing or working generally. In the absence of a conclusion that the employee had an impairment that substantially limited him/her from breathing or working generally, some courts have been unwilling to classify the employee as disabled within the meaning of the A.D.A.

These cases and others place asthmatic workers and their physicians somewhat in a Catch-22. If the report finds that the employee is totally impaired from working as a result of the respiratory disease, then the employee will not be deemed to be qualified to perform the essential functions of his or her position. Unless the disabled employee is otherwise qualified to do the job with reasonable accommodations, there will be no relief under the A.D.A. If the employee is deemed disabled from performing the particular job only because of specific workplace exposure, then it may be difficult to craft an accommodation request that a court will enforce. Obviously, merely recommending that the employee be transferred to or provided with an allergen or irritant-free workplace is likely to be rejected as simply too vague.

The lesson of all of this is that the treating or diagnosing physician must address three separate concerns in the report. First, the report must set forth sufficient medical test results or other factual findings to support a conclusion that the patient suffers from asthma or other respiratory disease that substantially limits the patient's ability to breathe and/or work. Second, the report must address whether the physician believes that the patient is capable of performing the essential functions of the job with reasonable accommodations. In order to be able to do this, the doctor must inquire into the essential functions of the position and must address how the patient will be able to perform those functions. Finally, the report must attempt to prescribe, with as much specificity as possible, the particular accommodations that the employee requires from the employer to be able to do his or her job. Recommendations as to regular break periods, the furnishing of desk-top air purifiers, the establishment of no-smoking zones, the allowance of workplace use of peak flow meters, spirometers, or other breathing apparatus, and the eradication to the extent possible of unnecessary irritants from the workplace are all the type of specific accommodations that a court is likely to enforce.

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Trends in Asthma Morbidity and Mortality, Rhode Island 1988–1997

Jay S. Buechner, PhD

Several recent national studies have presented evidence that the prevalence of asthma and the utilization of medical services to treat asthma have been increasing over the past decade or longer, both among the population as a whole and among young persons.[1,2,3] In addition, asthma death rates have been increasing among young persons.[3] Previous analyses on Rhode Island data showed that asthma prevalence rates in the state were higher than national rates as of 1996 and that the number of hospital inpatient discharges with asthma reported as an additional diagnosis increased between 1990 and 1995.[4,5] This report presents trend data on asthma-related mortality and hospitalizations in Rhode Island for the ten-year period from 1988 through 1997.

Methods

The Rhode Island death certificate file covers all in-state deaths plus deaths of Rhode Island residents occurring out of state and includes information on the decedent's demographics, underlying cause of death, and, since 1989, other conditions contributing to death. Rhode Island residents with asthma as the underlying cause of death and with any mention of asthma on the death certificate were identified based on the code for asthma (493) in the International Classification of Diseases, 9th Revision (ICD-9). Data for 1996 and 1997 are preliminary, as out-of-state deaths were not available.

Similarly, the statewide hospital discharge file includes patient demographics, principal diagnosis and additional diagnoses. Rhode Island resident discharges with asthma as the principal diagnosis and with asthma as an additional diagnosis were identified based on the code for asthma (493) in the International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM). Data on out-of-state hospitalizations of Rhode Island residents were not available for this analysis.

Crude and age-specific mortality rates and discharge rates were calculated using state population estimates from the United States Bureau of the Census.[6] Rates were produced for individual years and on an average annual basis for the five-year periods 1998-1992 and 1993-1997.

Results

Over the ten-year period examined, there were a total of 137 deaths in Rhode Island with asthma reported as the underlying cause. The mortality rate varied between 0.91 deaths per 100,000 population in 1994 and 1.80 deaths per 100,000 in 1992. (Figure 1) There was no indication of increasing mortality; the annual average rate decreased by 17% from the period 1998-1992 (1.49 deaths per 100,000) to 1993-1997 (1.24 deaths per 100,000).

There was also no evidence of increase in the rate of deaths with any mention of asthma on the death certificate during the nine years for which such data were available. Annual mortality rates for this definition varied between 3.14 deaths per 100,000 in 1997 and 5.20 deaths per 100,000 in 1992. (Figure 1) The decrease in average annual mortality rate from 1989-1992 (4.53 per 100,000) to 1993-1997 (3.78 per 100,000) was 17%, similar to the decrease in the underlying cause rate.

The rate of hospital discharges of Rhode Island residents with a principal diagnosis of asthma varied between 155.9 discharges per 100,000 in 1997 and 191.2 per 100,000 in 1993. (Figure 2) Again, there was no indication of increasing utilization for this condition; over 1998-1992, the average annual rate was 173.6 per 100,000, compared to 174.7 during 1993-1997. However, the discharge rate for patients with an additional diagnosis of asthma increased substantially during the decade, from a low of 118.5 per 100,000 in 1988 to a high of 435.2 in 1997, nearly a four-fold increase.

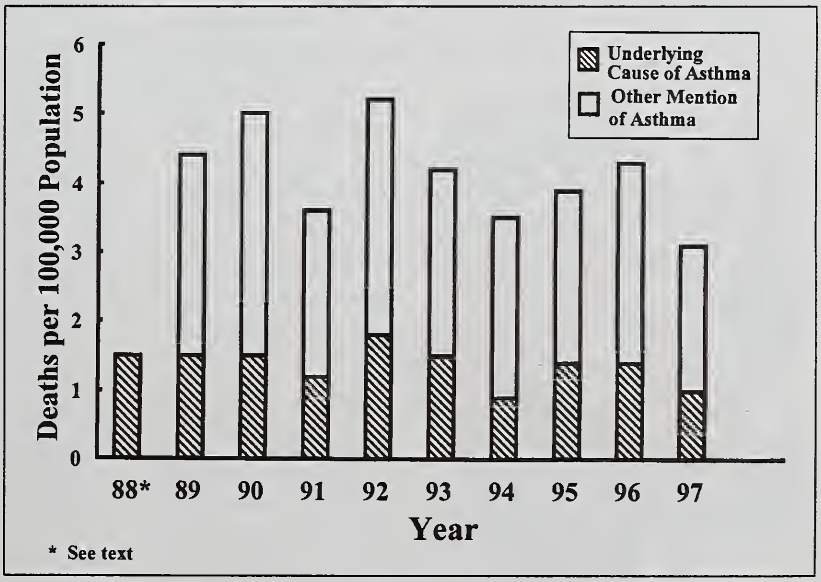

Figure 1. Asthma Mortality per 100,000 Population, by Year and Position of Asthma on the Death Certificate, Rhode Island, 1988 - 1997.

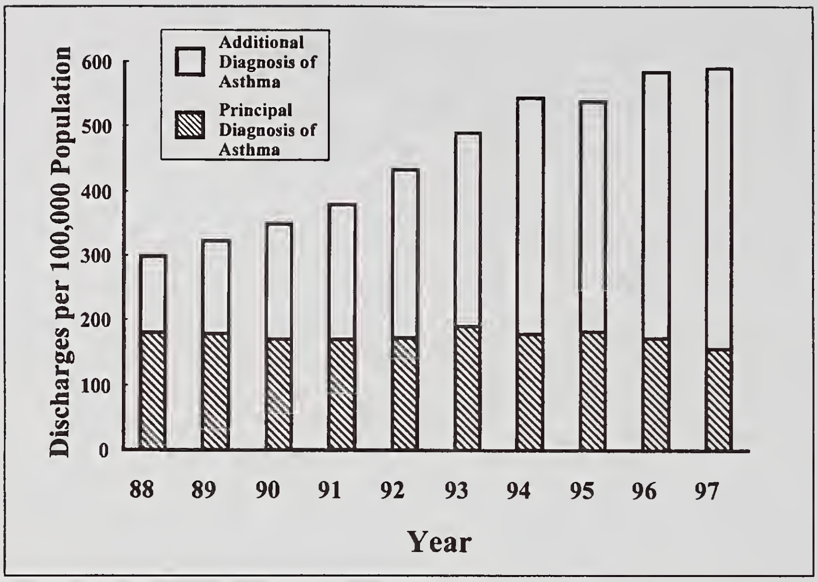

Figure 2. Hospital Discharges with Asthma per 100,000 Population, by Year and Position of Asthma Diagnosis, Rhode Island, 1988 - 1997.

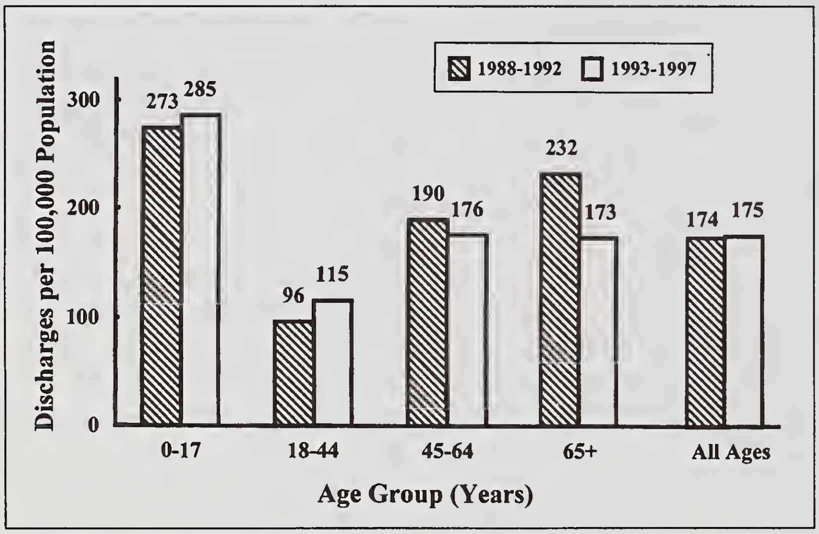

Figure 3. Hospital Discharges with Principal Diagnosis of Asthma per 100,000 Population, by Age Group and Grouped Years, Rhode Island, 1988-1992 and 1993-1997.

Although the overall discharge rate for patients with a principal diagnosis of asthma did not vary between 1988-1992 and 1993-1997, there was considerable variation in age-specific discharge rates. For the two younger age groups examined, ages 0 - 17 and ages 18 - 44, the discharge rate increased; among older persons the rate decreased, considerably (down 25%) for those ages 65 and older. (Figure 3) However, the rate of deaths with asthma as an underlying cause declined more greatly among younger persons (under age 45) than among older persons (ages 45 and older).

Discussion

Except for hospital discharges with an additional diagnosis of asthma, Rhode Island data show no evidence of an increasing burden of morbidity or mortality due to asthma. In fact, mortality rates, although based on small numbers of deaths, appear to have declined during the most recent decade for which data are available, especially among younger persons. The observed increase in hospital discharges with an additional diagnosis of asthma may be due to one or more of several possible effects, including the following: (1) increasing prevalence of asthma in the population, (2) increasing diagnosis of asthma by physicians, (3) increase in recognition of asthma as a complication for other conditions causing hospitalization, and (4) general increase in the number of additional diagnoses reported for hospital discharges in the state. Data are available only for the last-listed effect, and the observed increase can explain only a small portion of the increase in asthma-related hospitalizations. Examination of the other possible explanations will require additional information beyond what is currently available for the state.

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Pediatric Asthma Outpatient Case Presentation

Carla M. Martin, MD

INTRODUCTION

Jose (not his real name) is now a 37 month-old Latino male who had nearly fifteen hospital admissions and numerous urgent care and sick visits for asthma exacerbations during his first two years of life. The morbidity and mortality from asthma have been rising dramatically among urban children, with African-American and Latino children disproportionately affected. This case will address some of the factors health care providers have encountered in managing asthma. It also emphasizes the importance of multidisciplinary management of children with asthma and the socioeconomic variables that affect their care.

BIRTH HISTORY

Jose was born to a Spanish-speaking 19 year-old G2 P1-2 mother after an uncomplicated prenatal course and good prenatal care. She was Group B Strep positive and treated with a full course of ampicillin and cefazolin two and a half months prior to delivery.

The patient was born at 42 weeks by cesarean section secondary to failure to progress. Meconium was present and he was intubated and suctioned, given bag-valve-mask ventilation and blow by oxygen until given his heart rate, color and tone improved. The APGAR scores were 3 at 1 minute and 8 at 5 minutes. His birth weight was 4420 grams, birth length was 54 centimeters and head circumference was 37.25 centimeters. He was admitted to the Special Care Nursery for tachypnea.

HOSPITALIZATIONS AND ASTHMA MANAGEMENT

The patient became tachypneic several hours after delivery and was admitted for 2 days to the special care nursery and was ruled out for sepsis. He improved, was taken off antibiotics and went home with his mother.

The baby had been doing well at home, until 17 days of age when he became febrile and developed a cough. He was initially diagnosed with an otitis media and admitted the next day to the Pediatric Intensive Care Unit for respiratory distress. He was intubated during this admission and found to have Respiratory Synctial Virus bronchiolitis and pneumonia. He received his first nebulizer and IV steroid treatments, antibiotics and was discharged home on day 20 on prednisone and albuterol nebulizer treatments.

At 1.5 months of age, the patient was admitted again for pneumonia and RSV bronchiolitis. A murmur was noted during this admission and a cardiology evaluation including an echocardiogram was negative.

Following these admissions, the patient had approximately 13 more admissions for asthma exacerbations, the last one documented at 20 months of age. He also had approximately 15 urgent care, sick visits and follow-up visits for asthma, some of which led to admissions, and 11 emergency department visits for asthma exacerbations where he was not admitted. The patient also had several ED and urgent care visits for viral gastroenteritis and otitis media.

Abbreviations Used:

ED	emergency department
MDI	metered dose inhaler
PMD	primary medical doctor
RSV	respiratory synctial virus
VNA	Visiting Nurse Association

The patient's initial medical management consisted of albuterol nebulizer treatments. He would be given IV and then po steroids during and after his hospital admissions. Cromolyn sodium nebulizer treatments were added at approximately 9 months of age. When Jose was 11 months old, his mother was enrolled in the Pediatric Asthma Care and Education Program.

During an admission at 12 months of age, theophylline was added to his regimen. The pulmonary consult service noted that his mother was not administering his medicines correctly.

At 15 months of age, during his 12th hospital admission, Jose was started on beclomethasone MDI and continued on theophylline and his albuterol nebs. At 20 months of age, during an admission, the patient was taken off the theophylline and continued on the beclomethasone. Later that month, the theophylline was restarted during an admission. Several changes had been made to his regimen between his clinic visits. At age 21 months, the patient went to pulmonary clinic and was doing well. He was continued on the theophylline, changed from beclomethasone to flunisolide, and told to continue the inhalers. The providers had doubts about whether the mother was correctly administering the medications and arranged for a visiting nurse visit to ensure proper usage.

This regimen appeared to work well with minimal wheezing, and the patient was continued on it during his last documented pulmonary clinic visit, at age 22 months. Two weeks later, the patient had an urgent care visit for asthma exacerbation. He was continued on his medication regimen and started on prednisone. This was his last documented visit.

FAMILY HISTORY

The patient's family history was significant for asthma on the maternal side including his grandmother, several uncles and aunts. His parents and brother, who was four years older, had no significant past medical history and did not have asthma or any other medical problems.

FEEDING AND DEVELOPMENT

Review of the patient's feeding at his well-child visits deemed it appropriate. The patient was noted to be growing in the 50th percentile for weight which dropped to the 25th and then the 15th percentile at 12 months. His height dropped from the 25th to the 10th percentile at approximately the same time. His development was otherwise appropriate and his immunizations were up to date.

Allergen exposure, poverty, RSV, health care access and improper use of medications all contributed to this patient's asthma morbidity.

SOCIAL HISTORY

The patient initially lived with both of his birth parents and a brother, who was 4 years older. The primary caregivers were his parents but he was also cared for by his maternal and paternal grandparents. During his first 15 months, the patient's parents separated and his mother started a new relationship. The patient's father decided to move back to Puerto Rico and pressured the patient's mother to return with him. It is unclear if there had been any physical abuse in the household while the father lived at home, but the mother told the resident physician during Jose's 15 month-visit that she had a restraining order against Jose's father.

ALLERGENS

The VNA visited the patient's first home to evaluate possible environmental allergens. The apartment had cockroaches, rats and a wood burning stove. The parents denied smoking. The patient, his mother and brother moved to another apartment when the patient was 15 months old in an effort to avoid the allergens. The patient's brother, in daycare, often had upper respiratory infections.

DISCUSSION

Jose's case exemplifies the issues surrounding asthma management in children. Allergen exposure, poverty, RSV, health care access and improper use of medications all contributed to this patient's asthma morbidity.

The patient was exposed to multiple allergens at home including cockroach droppings, dust mites, rat droppings and a wood burning stove. In addition to the environmental allergens, he was diagnosed with multiple upper and lower respiratory infections. Despite his mother's efforts to limit the allergens, the patient continued to have exacerbations. The patient may have benefited from a visit to the Allergy and Immunology Clinic to evaluate specific allergens contributing to his asthma. A sweat test to rule out cystic fibrosis was done and was negative.

Sociodemographic and psychosocial variables are just as important. There is a well-known association between asthma morbidity and poverty. The patient's mother was young and came from a low income Hispanic household. Her perception of her child's health may have affected her response to it. So may her mental health, her social supports and the stress of her relationship with the patient's father. She had a restraining order against the patient's father but it was not clear whether she or the child were being abused. Social worker notes did not appear to address these. These stressors most likely contributed to Jose's health and led his mother to apply for disability coverage since she spent so much time caring for him. Health care providers must assess and respond to both the children's and the parent's stressors.

Jose had two admissions for RSV bronchiolitis. Given the association between RSV and future development of asthma secondary to bronchial endothelial damage, this probably also predisposed him to his multiple exacerbations. His mother frequently gave him the wrong medication regimen at home, despite receiving asthma teaching at the well-child and pulmonary clinics, during his hospital admissions and from the asthma education project. This emphasizes the importance of repetition in asthma education, especially with patients whose parents are primarily non-English speaking and who have low levels of schooling.

Jose did have adequate access to health care but did not have a consistent primary care provider. Instead, he was seen by multiple doctors. This may be partly due to the frequent hospitalizations, when he would miss his clinic visits, and also to the fact that he would often be assigned to a new doctor after his admissions. He also saw different doctors during his emergency department and urgent care visits. His mother was rarely able to identify the patient's PMD. The fact that Jose dropped to a lower percentile for height and weight is troubling. Behavioral issues affected by his asthma such as eating, sleeping and gastrointestinal symptoms do not appear to have been adequately addressed. Unfortunately, Jose was lost to follow-up and his last visit at the outpatient clinic was at the age of 22 months. His mother may have moved to Puerto Rico with him or may have taken him to a different clinic. As healthcare providers, it is frustrating when our patients are lost to follow-up, especially with a patient such as Jose who requires close management.

Over the past few years, we have seen increasing rates of morbidity and mortality from asthma among urban youth. As health care providers, we must take on the huge responsibility of devoting more time to educating our patients and addressing the social issues affecting their health, despite the constraints and time limitations placed on us.

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CME Background Information

This CME activity is sponsored by Brown University School of Medicine.

TARGET AUDIENCE

This enduring material is designed for primary care physicians.

CME OBJECTIVES

After completing this CME activity, the primary care physician will be able to meet the following objectives:

1. The physician will know the public health arguments, for and against, making asthma a "reportable disease" to the Health Department.
2. The physician will have a management plan, by visit, for treating a patient newly diagnosed with asthma; including
   * patient instructions as to self-care- instructions on exercise, household irritants and precautions to take
   * referral names/numbers/web site for support groups, asthma-related organizations
   * instructions for a "daily diary"
3. The physician will understand the specific management problems associated with inner-city urban children (allergen exposure, poverty, parents' stressors, improper use of medications, truncated professional care).
4. The physician will understand the practical isssues for working with the schools: whom to contact, what those contact people and cannot do. S/he will know about SBHC (school-based health clinics) and COZs (Child Opportunity Zones).
5. The physician will know the legal provisions for smoke-free schools.
6. The physician will understand the indications for prescribing antileukotriene modifiers.
7. The physician will know key environmental irritants, and the resources to call for additional information.
8. The physician will know Rhode Island's legislation for indoor air quality in public places.
9. A physician will be able to discuss the issue of early-prescription of steroids, knowing the arguments for and against.
10. A physician will know the rights of the worker with asthma under the Americans with Disabilities Act.

NEEDS ASSESSMENT

The need for this educational activity was supported by the Robert Wood Johnson Cooperative Actions for Health Program, which agreed to fund the issue.

ACREDDITATION STATEMENT

Brown University School of Medicine is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians.

This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education.

CREDIT DESIGNATION

Brown University School of Medicine designates this education activity for 2 hours in category 1 credit toward the AMA Physician's Recognition Award. Credit can be obtained by reading the issue and completing the quiz on pages 267-268. The estimated time for completion of this activity is 2 hours.

DATE OF ORIGINAL RELEASE

This issue was published in July 1999. This activity is eligible for CME credit through May 2000.

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AUTHOR DISCLOSER

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DISCUSSION OF INVESTIGATIONAL INFORMATION

The authors have stated there is no off-label or investigational use.

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TO OBTAIN CREDIT

To obtain credit, please submit answer grid and $25 fee to Office of Continuing Medical Education, Brown University. Respondents must receive a score of 70 or higher for credit.

CME QUESTIONS

Choose one response.

1. Making asthma a reportable disease would most likely lead to:
   a. reduced childhood prevalence
   b. improved therapeutic interventions
   c. competing disease registries
   d. a better disease definition
   e. lower mortality rates

2. Asthma affects almost_____ million people in the United States.
   a. 5
   b. 10
   c. 15
   d. 20
   e. 25

3. Which of the following statements is true?
   a. Patient education is of little importance.
   b. Most patients correctly use their metered-dose inhalers.
   c. Few other diseases mimic asthma.
   d. Peak flow alone should determine medication changes.
   e. An open dialogue between the physician and the patient is essential for successful treatment.

4. The use of inhaled steroids in children:
   a. may cause growth reduction in prepubescent ages
   b. is not approved by the FDA
   c. has no efffect on the frequency of exacerbations
   d. is associated with increased mortality
   e. should be reserved for exercise-induced broncho spasm alone

5. Leukotriene cause bronchoconstriction, increased vascular permeability and one of the following:
   a. increased ciliary function
   b. increased mucus secretion
   c. decreased edema formation
   d. decreased neutrophil recruitment
   e. increased IgE levels

6. Which of the following statements is true:
   a. Smoking is only allowed in school administrative buildings.
   b. Child Opportunity Zones (COZs) link health and social services to schools.
   c. Asthmatic children should not exercise.
   d. Only nurses can adminster inhalers at school.
   e. Asthma management plans aren't useful for school-aged asthmatics.

7. Environmental contributory factors for asthma include:
   a. indoor air pollution (cooking stoves)
   b. viral infections
   c. parental smoking
   d. house dust mites
   e. all of the above

8. To ensure coverage under the Americans with Disabilities Act, physicians must document which of the following:
   a. work impairment due to asthma
   b. medical confirmation of disease
   c. ability to perform job with reasonable accommodations
   d. a. & b.
   e. b. & c.

9. Asthma mortality in children has been associated with
   a. allergen exposure
   b. poverty
   c. health care access
   d. all of the above
   e. none of the above

10. Since_____, Rhode Island has banned smoking in school settings.
    a. 1998
    b. 1968
    c. 1975
    d. 1992
    e. 1984

11. Montelukast (singular®) has been proven to:
    a. increase as needed beta agonist use
    b. increase noctural awakening
    c. decrease quality of life measures
    d. increase FEV_1, and morning/evening PEFR
    e. none of the above

12. Enforcement of the smoke-free school act:
    a. has been comprehensive in most schools
    b. requires parents to write to the RI Department of Health
    c. starts with the school administration, then the super intendent and the school committee, if necessary
    d. is supervised by the RI Attorney General
    e. is not necessary

13. Indoor air quality in schools is protected by:
    a. OSHA
    b. RI Department of Labor and Training
    c. Environmental Health Division of the RI Department of Health
    d. RI Department of Health and Education
    e. all of the above

14. Physicians can help their school-aged asthmatics by:
    a. requesting school nurses to develop a written asthma management plan with the patient
    b. ignoring school policies and protocols concerning medications
    c. not informing parents, or school nurses, of problems in management
    d. sharing asthma education materials with their schools
    e. discouraging physical activity in school

15. Environmental causes of asthma include:
    a. air pollution
    b. house dust mites
    c. cigarette smoke
    d. pollen
    e. fungi

16. Smoking in the work place:
    a. is permitted in supermarkets, medical offices, and laundries
    b. is regulated by the Department of Health
    c. is allowed if accommodations are made for non-smokers
    d. none of the above
    e. all of the above

17. Rhode Island asthma data for the period 1988-1997 have shown:
    a. decreased overall asthma mortality
    b. increased overall asthma mortality
    c. decreased hospital discharges with an additional diagnosis of asthma
    d. increased overall hospital discharge rate for patients with a principal diagnosis of asthma
    e. a and c

18. Asthma can be confused with other disease states such as:
    a. foreign body obstruction
    b. panic disorder
    c. chronic tracheitis
    d. deconditioning
    e. all of the above

CME Registration Form

Circle one response for each question.

1.	a	b	c	d	e
2.	a	b	c	d	e
3.	a	b	c	d	e
4.	a	b	c	d	e
5.	a	b	c	d	e
6.	a	b	c	d	e
7.	a	b	c	d	e
8.	a	b	c	d	e
9.	a	b	c	d	e
10.	a	b	c	d	e
11.	a	b	c	d	e
12.	a	b	c	d	e
13.	a	b	c	d	e
14.	a	b	c	d	e
15.	a	b	c	d	e
16.	a	b	c	d	e
17.	a	b	c	d	e
18.	a	b	c	d	e

Print or type
Name ____________________
Address ____________________
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City, State, Zip ____________________
Phone (   ) ____________________
Fax(   ) ____________________
☐ Hospital ☐ Private Practice ☐ Resident ☐ Intern
☐ Other ____________

Deadline for submission

For credit to be received, please mail your registration with $25 fee, to: Office of Continuing Medical Education, Brown University School of Medicine, Box G-A2, Providence, RI 02912. Submit your answers no later than May 31, 2000.

Keep a copy for your files.

Retain a copy of your answers and compare them with the correct answers, which will be made available upon request, and receipt of submission requirements.

Evaluation

Please evaluate the effectiveness of the CME activity on a scale of 1 to 5 (1 being poor; 5 being excellent) by circling your choice.

	Poor Excellent
1. Overall quality of this CME activity	1 2 3 4 5
2. Content	1 2 3 4 5
3. Format	1 2 3 4 5
4. Faculty	1 2 3 4 5
5. Achievement of educational objectives:	
*Know the public health arguments, for and against, making asthma a “reportable disease” to the Health Department.	1 2 3 4 5
*Have a management plan, by visit, for treating a patient newly diagnosed with asthma.	1 2 3 4 5
*Understand the specific management problems associated with inner-city urban children.	1 2 3 4 5
*Understand the practical isssues for working with the schools: whom to contact, what those contact people and cannot do.	1 2 3 4 5
*Know the legal provisions for smoke-free schools.	1 2 3 4 5
*Understand the indications for prescribing leukotriene modifiers.	1 2 3 4 5
*Know key environmental irritants, and the resources to call for additional information.	1 2 3 4 5
*Know Rhode Island’s legislation for indoor air quality in public places.	1 2 3 4 5
*Discuss the issue of early-prescription of steroids, knowing the arguments for and against.	1 2 3 4 5
*Know the rights of the worker with asthma under the Americans with Disabilities Act.	1 2 3 4 5
6. This CME activity provided a balanced, scientifically rigorous presentation of therapeutic options related to the topic, without commercial bias.	1 2 3 4 5

Please comment on the impact that this CME activity might have on your management of patients.

____
____
____

Additional comments and/or suggested topics for future CME activities.

____
____

Rhode Island
Quality Partners, Inc.

Adult Immunization: Influenza and Pneumonia

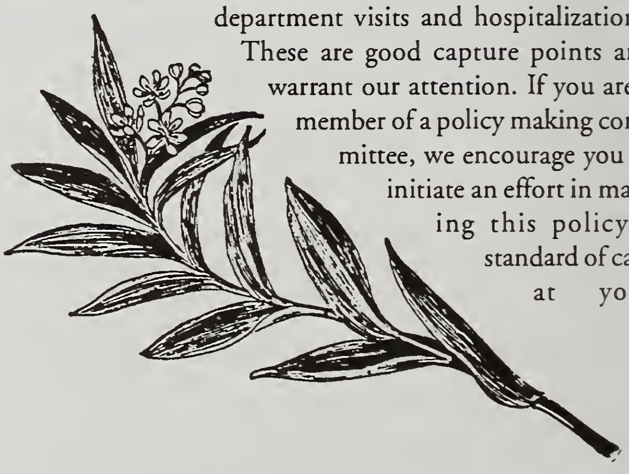
Raymond Maxim, MD

Once again it is time to prepare for the upcoming flu season which means that at Rhode Island Quality Partners (RIQP) we are also gearing up for our immunization program as well. Last year, we worked very closely with the Ocean State Adult Immunization Coalition to provide clinicians and beneficiaries with information regarding the influenza and pneumococcal vaccines. Medicare beneficiaries were encouraged to get both the flu and pneumococcal vaccines through a media campaign that depicted a blue hospital sign with a slogan * Wouldn*t you rather sleep in your own bed this winter?*. This generated significant interest among beneficiaries. The goal was to have patients ask their doctors for the vaccines instead of the providers having to remind patients of the need for immunization. We sent providers billing and vaccine purchasing information to help them prepare for the upcoming fall influenza season.

Last year, in addition to our usual informational mailings we did something different. We sent to physicians listed as Internal Medicine, Family Practice, General Practice, and Geriatrics the rates for pneumococcal vaccine in beneficiaries that we could identify as one of their patients. The results were both disturbing and encouraging. The discouraging information was that the rates averaged less than 20% for patients having received a pneumococcal vaccine in the previous 8 years. The few practitioners with high rates had very small numbers of patients. As a rule, the primary care practitioners performed very poorly in administering an effective preventive service. I must admit my own practice did not perform any better than others. My personal rate for pneumococcal immunizations was only 26%. As you can imagine, this was extremely disappointing to me and my staff. With that information we chose to change the practice to include standing orders for both influenza and pneumococcal vaccinations. These standing orders are not limited to those patients that have appointments. Anytime a patient comes to the office whether it be for a billing problem or to ask for a prescription refill they are offered an appropriate vaccine in order to capture every immunization opportunity.

The good news is that the response from other practitioners was similar to my own. I received letters and E-mail from colleagues expressing their surprise and announcing their determination to review their practice patterns in order to improve their performance. This is an example of benchmarking and how it can help physicians improve their performance. This type of feedback is essential but can be expensive and time consuming for an individual practice to gather. RIQP will be providing similar information about influenza and pneumococcal vaccines this year. We hope that you will find this information helpful in caring for your patients.

To improve performance as a whole for the state of Rhode Island from the current level of 50% of eligible Medicare beneficiaries receiving immunization for influenza we must address those populations that are at risk and are difficult to reach. Suggestions from other physicians have been standing orders for immunizations in the office that make it routine to inquire about immunization status. For those of you who are medical directors of nursing facilities, consider using standing orders on admission for pneumococcal immunization and seasonal orders for flu vaccine. With the implementation of the prospective payment system, it is in the institutions* best interests to be proactive in preventing hospitalizations. Standing orders that have been used successfully in other institutions are available and I would be happy to share those with you. Other opportunities pointed out by physicians that have contacted me are emergency department visits and hospitalizations. These are good capture points and warrant our attention. If you are a member of a policy making committee, we encourage you to initiate an effort in making this policy a standard of care at your



hospital. There are many hospitals outside the state of Rhode Island that have had successful programs and at least one hospital in Rhode Island who has begun a similar program.

As usual, I seek feedback from you regarding these issues or any others pertaining to the quality of care given our Medicare beneficiaries.

Raymond B. Maxim, MD, is Associate Clinical Coordinator, RIQP; Clinical Instructor, Brown University School of Medicine: and staff physician, Roger Williams Medical Center.

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The author assumes full responsibility for the accuracy and completeness of the ideas presented. This article is a direct result of the Health Care Quality Improvement Program initiated by the Health Care Financing Administration, which has encouraged identification of quality improvement projects derived from analysis of patterns of care, and therefore required no special funding on the part of this Contractor. Ideas and contributions to the author concerning experience in engaging with issues presented are welcomed.

SEQUELAE TO THIS ISSUE: WORKSHOPS

The Robert Wood Johnson CAHP grant has underwritten not only this issue of *Medicine & Health/Rhode Island*, but post-issue workshops. That post-issue money will go towards grand rounds in hospitals, inservice sessions for school nurses, and workshops in inner-city schools. For more information, contact Celia Gomes-McGillivray, MPH, past president of the Rhode Island Public Health Association, phone: 459-5784; e-mail: cgm@ids.net.

Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	July 1998	12 Months Ending with July 1998		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	226	3,131	316.2	3,965.0
Malignant Neoplasms	213	2,520	254.5	6,873.5**
Cerebrovascular Diseases	50	655	66.1	739.5
Injuries (Accident/Suicide/Homicide)	39	346	34.9	7,065.5**
COPD	34	464	46.9	350.0

Vital Events	Reporting Period		
	January 1999	12 Months Ending with January 1999	
	Number	Number	Rates
Live Births	914	13,254	13.4*
Deaths	973	9,741	9.8*
Infant Deaths	(6)	(97)	7.3#
Neonatal deaths	(4)	(73)	5.5#
Marriages	261	7,480	7.6*
Divorces	220	3,112	3.1*
Induced Terminations	509	4,616	348.3#
Spontaneous Fetal Deaths	10	837	63.2#
Under 20 weeks gestation	(1)	(750)	56.6#
20+ weeks gestation	(9)	(87)	6.6#

* Rates per 1,000 estimated population # Rates per 1,000 live births

**Excludes one death of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 990,225

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

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Heritage: Asthma Reports

January 1900

In "Some Observations On Bright's Disease," Herbert Terry, MD, relayed the observations of Dr. Whitfield, who cautioned colleagues to check patients' urine - both when they suspected renal insufficiency, and when they didn't. In two cases asthma was symptomatic of Bright's Disease. A painter who had come to [Dr. Whitfield's] office had difficulty breathing and thought he had a cold. Within the year the painter died. In another case, a carpenter had "bronchitis of a rather long duration, and with more than the usual bronchial rasp." Checking the carpenter's urine, Dr. Whitfield diagnosed Bright's disease. [Dr. Whitfield also linked Bright's disease to ulcers, nosebleeds, diarhhea, cirrhosis, pleurisy, epilepsy, migraine, and insanity.]

January 1934

In "The Rhinological Treatment of Asthma," Jay N. Fishbein, MD, declared, "It is only of comparatively recent times that the nose and throat have been seriously considered as a potential factor in the causation of asthma." Fifty years ago Dr. Voltolini had reported curing asthma after a nasal operation - spurring "rhinologists everywhere" into "rushing blindly into nasal surgery as a cure-all for bronchial asthma." From Fishbein's vantage, this enthusiasm "doomed Voltolini's discovery." A few patients emerged cured; many emerged worse, after a "needless sacrifice of intra-nasal tissue."

Other advances: Dr. James Adam (Glasgow, 1900) posited a dual causation: a toxin combined with a lesion in the respiratory tract (generally the nose) would lead to asthma. Dr. Burton Hazeltine (Chicago, 1900) posited that asthma was an anaphylactic effect of an allergy. The English surgeon Sir Dundas Grant (1927) posited an "asthma-genetic zone" in the ethmoid region of susceptible people.

Empirically, a 1929 study at the University of Pennsylvania of 200 cases showed that 173 had positive sinus disease.

Dr. Fishbein argued against radical surgery ("nasal and sinus operations should not be performed primarily for the cure of asthma"). He urged physicians to wash away nasal secretions ("tamponage augmented by diathermy"), suggesting colloidal silver Dowling tampons. The patient would wear fastened to his forehead (by a letter-carrier strap) a tin block with strips of diathermy tape attached to a 2' by 8' strip of rubber. If the patient was hyper-sensitive to dust or food, the physician should desensitize the patient. If no hyper-senstivity was found, the physician should initiate non-specific (Protein shock) treatment - the parenteral injection of a non-specific protein. Dr. Fishbein used a variety of vaccines (typhoid, Colon bac, Van Cotts combined Bacterial Vaccine, Sherman's Vaccine, Milk Injections).

Of 170 patients (ages 5 to 60+), 42 had complete relief with this treatment. As for etiology, 128 patients had a relative with hay fever or asthma; 119 themselves had hay fever. In 84% of cases, Dr. Fishbein found a nasal pathology; in 47%, an infection of the ethnoid sinuses.

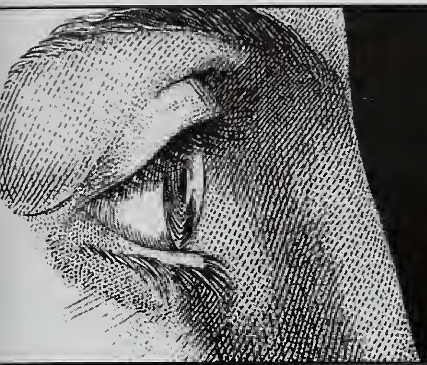
March 1934

In "The Specific Treatment of Asthma in Children," William P. Buffum, MD, discussed the "offending substance" - an inhaled (pollen, animal dander, dust) or ingested atopen. He urged colleagues to test patients, then remove the offending substance. He conceded, though, the difficulty of this "sort of shot-gun" approach of trying different exclusions, documenting sensitivity, then administering vaccines. Many patients would show no one clear sensitivity, particularly "the small group of malnourished children with severe asthma."

He isolated house dust (identified in 1922 by Cooke as an atopen) as a key irritant: "half of all asthmatics are sensitive in some degree by skin test to a preparation of house dust." Dr. Buffum injected a stock vaccine for house dust intravenously in 21 children; 11 improved. Conceding the difficulty of revamping a child's environment, Dr. Buffum occasionally recommended the family move. "One form of treatment which is useful occasionally, especially in severe cases, is a change of residence...if the patient moves, he will be better."

In "Asthma Study in Children," Reuben Bates, MD, and Stanley Freedman, MD, reported on 108 patients seen in the Children's Asthma Clinic of Rhode Island Hospital, 1930-34. The children, ages 3- 13, had suffered from 6 months to 11 years (the average was 3.5 years). Thirty-two percent had a history of infantile eczema; 59%, a history of atopy.

The authors listed common triggers: cat hair, rabbit hair, chicken feathers, June grass, orchard grass, ragweed, egg yolk, egg white, tomatoes, strawberries, salmon, house dust. As for etiology, they linked asthma to a variey of diseases: pertusses, measles, scarlet fever, pneumonia, influenza. As for treatment, they advocated tonsillectomy, even though they couldn't explain why it worked. "Why this surgical operation benefits asthmatics we are unable to explain, unless it is due to non-specific shock which temporarily benefits the allergic state."



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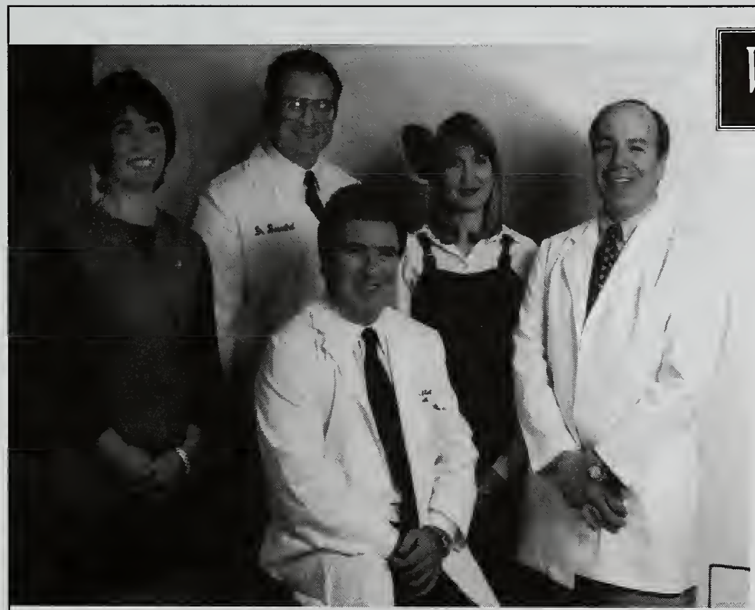

Photo: Peter Ventrone

Seated: Dr. Nicholas Iannuccilli; Standing, left to right: Dr. Kathleen Staudinger; Dr. Charles Rosenthal, Dr. Karen Ackerman and Dr. Daniel Druckman; Not pictured are Dr. Paul Hessler and Dr. Rodolfo Zaldivia

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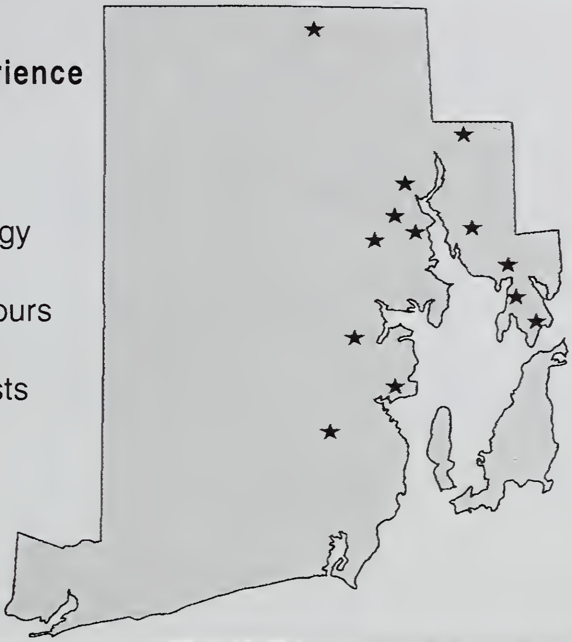

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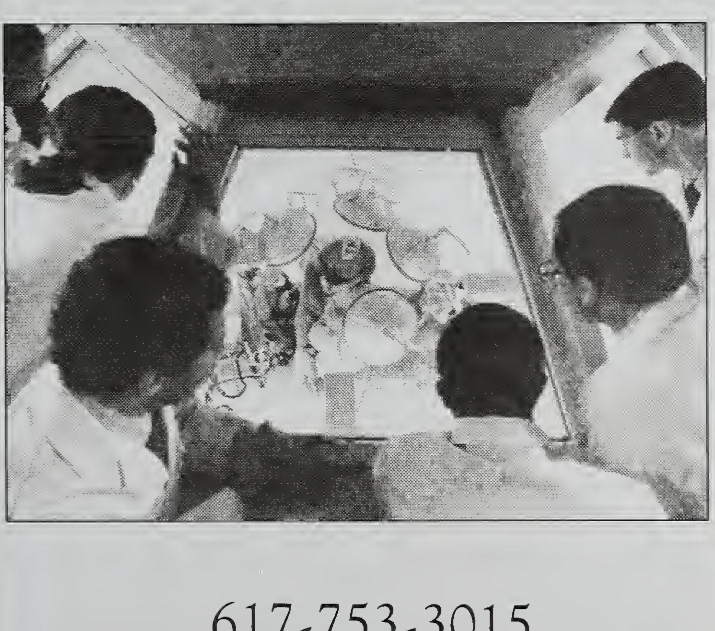

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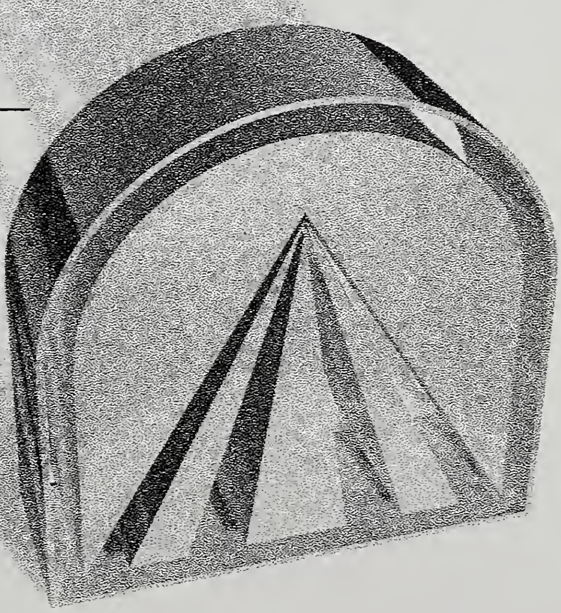

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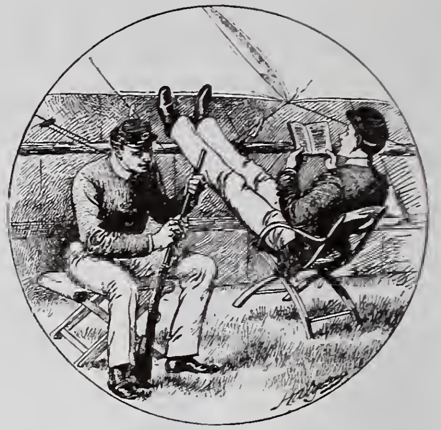
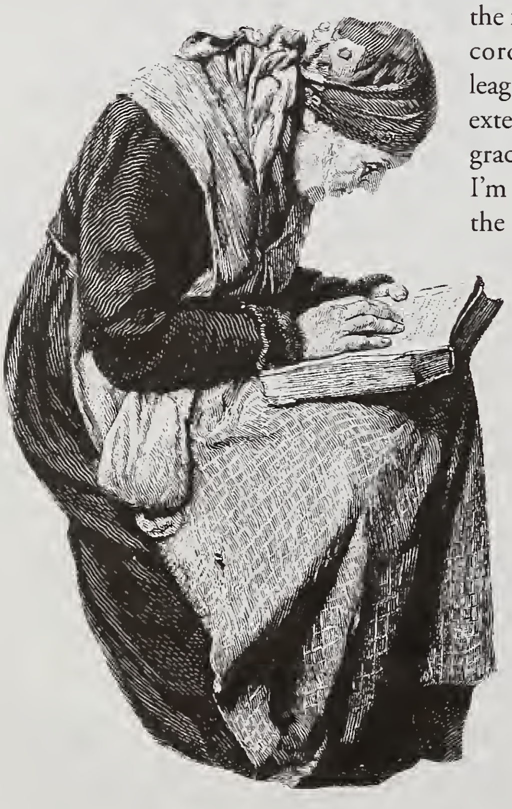


Medical Etiquette: Pro and Con

Etiquette is the social grease that smoothes our interactions. Male doctors are often held to a lower standard than female because they are perceived as important, or busy, or burdened with the weighty matters of their hurt and dying patients, hence distracted. Female doctors are held to a higher standard than their male counterparts, just as women are in the wider society.

We have different rules for different situations. We behave one way with our patients, another with our colleagues and yet another with students, administration and the outside world. None of us likes to observe rudeness and we all certainly detest receiving it. Etiquette is, I believe, discussed too little with medical students and housestaff. Etiquette reflects training but also sensitivity. One is limited by not having sensitivity or training. Rules of interaction are culturally specific and must be learned. Occasionally one learns to be impolite. Young doctors sometimes learn to be rude, equating rudeness with importance. The chief, who stands as a role model and demands and sometimes demeans, creates an atmosphere in which nascent physicians confuse the notions of "demanding" with "commanding" respect.



There are certain situations in which doctors interact with their colleagues that evoke rudeness. Perhaps most common is the junior physician, sometimes a house officer, more commonly a fellow, called in as a consultant, who offers an opinion that is not the desired one. Although we frequently request second opinions, we often don't really want them. We want the first opinion repeated. Getting discordant opinions from junior colleagues makes most of us bridle to some extent, hopefully covered up with good grace. "Yes, that's very interesting, but I'm sure that's bone wax leaking from the sternum, not pus." A less nice response is, "I've seen this hundreds of times and I know it's not pus. Get your boss!" The best response of course, hard to muster when in a hurry, the case complex and the patient is doing poorly, "Well, I don't think you're correct but how can we figure out which one of us is correct?"

We often slight our colleagues by not contacting them to update them on their patients, in both the in and outpatient arenas. I know we suffer from this in my office (due to staff shortage), but we try to update our colleagues on important issues. Good etiquette is usually good medicine.

An interesting area of conflict I've seen is in the use of limited facilities where two group physicians sign up for the same room for consecutive time slots, but the first group tends to run late, perhaps even to run late on purpose and instead of starting early or watching the clock to end on time, tells the second group to find another room or time. "We won't finish on time unless we want to." "Your group isn't ready anyway." "Why don't you find another room?" What drives this rudeness? Certainly it's not good for the physician groups to resent each other. This rudeness undoubtedly filters down to junior physicians.

At a distinguished medical school in the South there was consideration given to establishing a course in medical etiquette. While it may sound surprising, one of the hospitals where I am on staff sent a memorandum not long ago requesting that attending physicians behave civilly on the wards, not shout at nurses or throw things.

Proper etiquette is not always good. Allowing bad medical care for fear of violating the usual social rules is wrong. Most of our daily interactions go smoothly because we follow the "rules." When we don't there should be a reason that centers on patient care or education, not physician ego.

– Joseph H. Friedman, MD

The Dutchman Who Discovered a Vast New World

Mortal life, prior to the 17th Century, was said to be confined to those plants and creatures that could be readily seen or touched. Isolated observers may have recklessly suggested the existence of animalcules too small to be seen; but few believed this bizarre notion since it was patently contrary to the early words of *Genesis*. And then there were speculations concerning intangible forces emanating from swamps and putrefactive matter, poisonous forces called miasma; but these noxious substances were not considered to be alive.

A succession of elaborate theories were constructed to explain such inexplicable events as contagious disease, but without acknowledgement of the invisible world of microbial life, these various conjectures inevitably faltered and the dynamics of communicable infection went unexplained. The existence of an entire kingdom, an invisible domain, had yet to be demonstrated to a skeptical world.

Something was needed, therefore, to view the broader tapestry of life, something to demonstrate that there was life though it be invisible to the unaided eye. That something was an instrument called a microscope, a means by which a vast new world of fantastic denizens might then be inspected, measured and prodded.

The scientific community of the 17th Century was awash with theories to explain all manner of natural and pathological phenomena. What was needed, ultimately, was an intensely curious human, unhampered by a rigidly scholastic education, unburdened by inherited preconceptions; a person who was scrupulously honest and blessed with an uncontaminated, childlike enthusiasm to observe things, not as they should be but as they are.

Such a person was Antoni Leeuwenhoek, born in 1632 in Delft, Holland, the fifth child of a basket weaver. His father died when Antoni was five and he was raised by a maternal uncle, a town clerk.

Antoni was an undistinguished student learning little more than the rudiments of mercantile arithmetic and a smattering of commercial law but no Latin or any of the contemporary languages of Europe other than his native tongue. His literary powers were limited to terse, declarative Dutch.

At age 16 he moved to Amsterdam embarking on a career as a bookkeeper to a draper. He married shortly thereafter but within 12 years his wife had died. A second marriage in 1671 enabled him to assume certain civic responsibilities [chamberlain and wine gauger of Delft] thus freeing some of his time to pursue his private interests.

Antoni's new interests included the principles of navigation, the expanding science of astronomy and the arcane art of lens grinding. His catholic curiosity ranged from the mechanisms of barometers to the forces governing the movement of the winds.

Single-lens microscopes had been constructed in the 17th Century but were so technically poor that little new information at the cellular level was forthcoming. Antoni's lenses, however, allowed magnifications exceeding 200-fold without appreciable loss of resolution.

The year was 1673 and England and Holland were engaged in a great naval war. Despite this a Delft physician, Reinier de Graaf, wrote a letter to London stating: " . . . that one Mr. Leeuwenhoek hath lately contrived microscopes excelling those that have hitherto been made . . ." The letter was addressed to the Secretary of an astonishing organization called the Royal Society for the Improving of Natural Knowledge. The Society was begun in 1660 as a clearing house for new scientific information. It sought out correspondents throughout the world to share openly their new discoveries with like-minded scholars. The correspondence was published in *Philosophical Transactions,* now the world's oldest active scientific periodical.

For the next 50 years Leeuwenhoek submitted a flood of letters to the Royal Society, each filled with novel observations. His contributions were duly translated to Latin and then widely disseminated. In a lifetime of some 90 years this unlettered, essentially uneducated civil servant defined the field of microscopy, fathered the science of microbiology, and made a multitude of discoveries concerning the structure and function of plant and animal tissue. Rarely in the history of science have one person's contributions been of such monumental importance.

In a letter to the Royal Society in 1675, Leeuwenhoek provided the first known description of heretofore invisible creatures [including protozoa and bacteria] busily moving about in pond water. The Fellows of the Royal Society did not initially accept these findings until Robert Hooke was able to verify their existence. Leeuwenhoek's detailed observations allowed him to explain how these creatures were able to move [by cilia], how they reproduced themselves [by binary fission] as well as their ubiquity and variety of forms.

Human reproduction had been construed as the mystical union of male and female elements. The male contribution, semen, was said to contain an ill-defined spirit, a vital heat [*calor innatus*] which was derived from the male heart and which was capable of initiating life. Leeuwenhoek studied semen under his microscope and discovered instead myriads of "living animals." Despite his squeamishness in broaching a subject of such delicacy, he nonetheless proceeded to provide a detailed morphology of sperm, not only in humans but in a broad representation of vertebrate life.

His later studies included the structure of ovaries, how plants propagate, the detailed anatomy of insects, the micro-anatomy of blood cells and the structure and function of capillaries. His reports were brief, focused, unambiguous - and always in vernacular Dutch. Yet his contributions were fully appreciated by scientists throughout the world. He was unanimously elected a Fellow of the Royal Academy, his diploma carefully inscribed in Dutch rather than the customary Latin.

Leeuwenhoek was the model of a Netherland bourgeoisie. He travelled little, lived modestly, trained no disciples and wrote no texts other than than the hundreds of letters to the Royal Society. It is a sad irony that both of his wives as well as five of his six children succumbed to infectious fevers caused, no doubt, by some of the insidious microorganisms which his researches had caused to be known.

– Stanley M. Aronson, MD

Brown University School of Medicine: Class of 1999

Stephen R. Smith, MD, MPH, Alexandra Morang, Hilary Sweigart, and Janice Viticonte

On May 31, 1999, 85 men and women received the Doctor of Medicine degree from Brown University representing the 25th class of physicians graduated from that institution in this century. If this class follows the pattern of preceding classes, approximately 14% will eventually enter the practice of medicine in the State of Rhode Island. Of the 1738 physician graduates of previous classes, approximately 232 (13%) are currently licensed to practice in Rhode Island.

The purpose of this article is to introduce the graduates of the M.D. Class of 1999 to the physician community in Rhode Island, as many will be your future professional colleagues.

Table 1. Demographic Characteristics of the M.D. Graduates of the Brown University School of Medicine Class of 1999

Sex		
Male	46	54%
Female	39	46%
Race		
White	41	48%
Asian American	34	40%
African American	5	6%
Hispanic	2	2%
Foreign National	2	2%
Puerto Rican	1	1%
State of Residence		
New York	22	26%
Rhode Island	10	12%
California	8	9%
New Jersey	8	9%
Massachusetts	5	6%
Connecticut	5	6%
Pennsylvania	3	4%
Arizona	2	2%
New Hampshire	2	2%
Maryland	2	2%
Indiana	2	2%
Illinois	3	4%
Mississippi	2	2%
Other States	10	12%
Other Countries	1	1%

A PORTRAIT OF THE CLASS OF '99

Forty-six graduates were men (54%) and 39 were women (46%). The racial/ethnic composition of the class (Table 1) shows a slightly higher proportion of students from Asian American backgrounds (40%) than the previous year. Almost half of the students in the first four undergraduate years of the eight-year Program in Liberal Medical Education (PLME) are Asian American. Seven percent of the graduates are members of minority groups underrepresented in medicine (5 African Americans and 1 mainland Puerto Rican) as defined by the Association of American Medical Colleges (AAMC). Two other graduates are Hispanics but neither Mexican American nor mainland Puerto Rican and thus not counted in the AAMC definition. This number is lower than the 14% URM reported for last year's graduates. For the past five years, about 20% of freshmen college PLME students have been from underrepresented minority groups. The percentage of

Abbreviations Used:

AAMC	Association of American Medical Colleges
EIP	Early Identification Program
PLME	Program in Liberal Medical Education
URM	underrepresented minority

Fig. 1

Specialty Choices of the Brown University School of Medicine Class of 1999

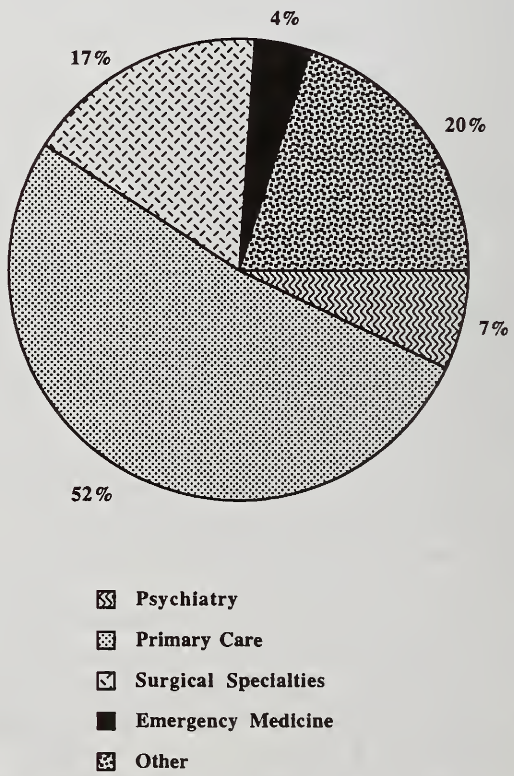


Table 2. Specialty Choices of the MD Graduates of the Brown University School of Medicine Classes of 1995–99

	1995		1996		1997		1998		1999	
Specialty Choice	No.	Percent	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Primary Care, Total	41	58%	53	60%	52	59%	46	61%	44	52%
Internal Medicine, total	18	25%	25	28%	16	18%	18	24%	15	18%
Categorical Medicine	15	21%	18	20%	11	13%	13	17%	14	16%
Primary Care Medicine	3	4%	7	8%	5	6%	5	7%	1	1%
Pediatrics	14	20%	10	11%	10	11%	14	18%	7	8%
Family Medicine	4	6%	13	15%	18	20%	10	13%	12	14%
Medicine/Pediatrics	0	0%	1	1%	3	3%	1	1%	2	2%
Obstetrics & Gynecology	5	7%	4	5%	5	6%	3	4%	8	9%
Surgery	7	10%	4	5%	6	7%	5	7%	9	11%
Surgical Subspecialties, total	9	13%	8	9%	10	11%	6	8%	5	6%
Ophthalmology	3	4%	2	2%	2	2%	2	3%	0	0%
Orthopedics	3	4%	4	5%	4	5%	1	1%	3	4%
Neurosurgery	0	0%	0	0%	1	1%	0	0%	0	0%
Urology	3	4%	0	0%	0	0%	1	1%	1	1%
Plastic Surgery	0	0%	1	1%	2	2%	2	3%	1	1%
Otorhinolaryngology	0	0%	1	1%	1	1%	0	0%	0	0%
Dermatology*					1	1%	1	1%	0	0%
Emergency Medicine	0	0%	3	3%	9	10%	5	7%	3	4%
Psychiatry	3	4%	2	2%	3	3%	2	3%	6	7%
Neurology	2	3%	4	5%	2	2%	1	1%	0	0%
Transitional and Preliminary	0	0%	6	7%	0	0%	1	1%	13	15%
Institutional Specialties, total	8	11%	2	2%	5	6%	5	7%	1	1%
Anesthesiology	3	4%	0	0%	1	1%	1	1%	0	0%
Pathology	0	0%	1	1%	0	0%	0	0%	1	1%
Rehabilitation Medicine	1	1%	1	1%	0	0%	0	0%	0	0%
Radiology and Radiation Oncology	4	6%	0	0%	4	5%	4	5%	0	0%
Delaying Residency	1	1%	6	7%	0	0%	4	5%	4	5%
Totals	71	100%	88	100%	88	100%	76	100%	85	100%

*Prior to 1997 1st-year match in dermatology not possible

underrepresented minority students in the first three years of the medical school is 17%.

Ten graduates are residents of Rhode Island. They came from eight different communities in the state, with three from Providence, and one each from Central Falls, Cranston, Johnston, Lincoln, North Providence, Smithfield, and Warwick. Their high schools also reflect this diversity: Johnston, Lincoln, Moses Brown, N. Smithfield, St. Raphael Academy, St. Mary's Academy, the Lincoln School, and Toll Gate.

The M.D. Class of 1999 reflects the growing proportion of students from the PLME: 50 graduates (59%) came through that route. The second largest cohort of students (16 graduates) came through the combined Brown-Dartmouth Medical Education Program in which students spend their first two years of medical school at Dartmouth, then transfer to Brown for the final two years.

The medical school entered into special agreements with several postbaccalaureate premedical programs (Brown University, Bryn Mawr College, and Columbia University) shortly after the PLME was inaugurated. Students from these programs decided upon a career in medicine after completing college. Typically, they have been engaged in other careers for several years following college. The goals in establishing this new route of admission were to maintain a rich diversity in the student body by admitting students who were older and who had different academic and life experiences as well as rounding out the total class size to compensate for the expected attrition from the PLME.

Postbaccalaureate students represented 11% of the graduates. Of the nine postbaccalaureate students, five came through Bryn Mawr College, one from Brown, and three from Columbia University.

Among the remainder of the class, four students were part of the Early Identification Program (EIP), one each from Providence College and the University of Rhode Island, and two from Tougaloo College. EIP students are offered provisional admission to the medical school during their sophomore year at their respective undergraduate colleges. The remaining six graduates entered medical school through either the MD/PhD program, advanced transfer, or the Brown Avenue, the latter being available to Brown students not in the PLME.

Brown University was the most common undergraduate college (53 graduates). Harvard, Tougaloo, Wesleyan, and Yale were tied for second with two members each from the Class of 1999.

The most common undergraduate major was biology (including subdisciplines such as neural sciences and microbiology), with 61 % of the class selecting that as their undergraduate field of study. Science majors taken together (including psychology) accounted for 74% of all majors, while 16% majored in the humanities and 11% majored in the social sciences. Among the humanities majors, English was the most common choice, while health and society was the most popular choice among the social science majors.

WHERE THEY ARE GOING

Internal medicine remained the most frequently selected specialty (15 students), although family medicine placed a very close second (12 students). (Table 2)

The proportion of the class entering specialties in primary care dropped to 52% of the class from 61% in the previous year. This includes the fields of internal medicine, pediatrics, family practice, medicine/pediatrics, and obstetrics and gynecology. (Figure 1) The number of graduates who will eventually practice primary care after completing their graduate medical education training will be smaller than the 52% reported here, if the past is any indication. An analysis of longitudinal data collected by the AAMC of the graduates of the classes of 1987-1991 reveals that over half of the graduates

Table 3. BROWN UNIVERSITY SCHOOL OF MEDICINE CLASS OF 1999 RESIDENCY POSITIONS

NAME OF GRADUATE	HOSPITAL NAME/MEDICAL SCHOOL AFFILIATION	SPECIALTY
Devon Addonizio	Long Island College Hospital / SUNY-Brooklyn	Pediatrics
Arshad Ahsannudin	University Health System Eastern Carolina/East Carolina Univ	Pathology
Richard Alweis	Mt. Auburn Hospital / Harvard Medical School	Medicine
Brenna Anderson	University Health Center-Pittsburgh / Univ.of Pittsburgh	Ob/Gyn
Curt Beckwith	Brown University Internal Medicine Residency	Medicine
Eileen Birmingham	UC San Francisco	Pediatrics-Primary
Deborah Borne	UC San Francisco	Family Practice
Jeffrey Brown	Highland Hospital / University of Rochester	Family Practice
Gloria Browne	Martin Army Community Hospital/Fort Benning, GA	Family Practice
Tira Bunyaviroch	Rhode Island Hospital / Brown University	Surgery-Prelim
Gerry Carino	Brown University Internal Medicine Residency	Medicine
Larry Chan	Kaiser Permanente-Los Angles / UCLA	Medicine
Nancy Chan	New England Medical Center / Tufts University	Psychiatry
Michelle Chen	Lutheran General Hospital / Univ of Chicago-Pritzker	Pediatrics
George Cushing	Cambridge Hospital / Harvard Medical School	Transitional
	Mt. Auburn Hospital / Harvard Medical School	Radiology
Justin Dominick	UC San Diego Medical Center	Medicine-Prelim
	UCLA Medical Center	Neurology
Gerard Gagne	Brown University	Psychiatry
Neel Gandhi	NY Presbyterian Hospital / Columbia	Medicine-Primary
Graham Gardner	Beth Israel Deaconess Medical Center / Harvard Medical School	Medicine
Stephen Gilheeney	NY Presbyterian Hospital / Cornell University	Pediatrics
Jon Grant	University of Minnesota Medical School	Psychiatry
Janelle Guirguis-Blake	UC Davis Medical Center	Family Practice
Deborah Gutman	Rhode Island Hospital / Brown University	Emergency Med
Omid Hajiseyedjavadi	NYU Medical Center	Surgery
Carman Hall	Univ. of Texas Medical School-Houston	Family Practice
Wendy Hall	Oregon Health Sciences University	Ob/Gyn
Christopher Healey	Univ. of Vermont / Fletcher Allen	Surgery
Ira Helenius	Mt. Sinai Hospital / Mt. Sinai School of Medicine	Medicine-Prelim
Kenneth Hsiao	Emory University School of Medicine	Urology
Linda Huffer	San Antonio Uniformed /Ft. Sam Houston	Medicine
Christine Huo	Stanislaus Health Services / UC-Davis	Family Practice
Andrew Kalinsky	Brown University Internal Medicine Residency	Medicine-Prelim
	Rhode Island Hospital / Brown University	Radiology
Patrick Kang	Brown University Internal Medicine Residency	Medicine-Prelim
	St. Lukes-Roosevelt / Columbia	Radiology

Aftab Karim	Univ. of Arkansas-Little Rock	Surgery-Prelim
	Univ. of Arkansas-Little Rock	Neurosurgery
Karen Kim	Beth Israel Deaconess Medical Center / Harvard Medical School	Medicine-Prelim
	SUNY Health Science Center-Brooklyn	Dermatology
Melisa Lai	Brigham & Women's Hospital / Harvard Medical School	Emergency Med
Phillip Lai	NYU Downtown Hospital / New York University	Medicine-Prelim
	George Washington University Hospital	Ophthalmology
Mark Lee	UMDNJ-RW Johnson-Piscataway	Medicine
Gretchen Lockard	Univ. of Washington Affiliated Hospitals	Family Practice
Felix Lui	Berkshire Medical Center / University of Massachusetts	Surgery
Maria Lui	Northridge Hospital Medical Center / UCLA School of Medicine	Family Practice
Jennifer Martinelli	Boston University Medical Center	Family Practice
Erika Mitchell	University Hospitals of Cleveland / Case Western Reserve Univ.	Orthopedic Surgery
Landis Mitchner	Butler Hospital / Brown University	Psychiatry
Beata Napiokowski	Hospital of the University of Pennsylvania	Ob/Gyn
Ka-Kei Ngan	Roger Williams Hospital / Boston University	Medicine-Prelim
	University of Connecticut	Radiology
Garey Noritz	MetroHealth Medical Center / Case Western Reserve Univ.	Med/Peds
Susan Oliver	University of Massachusetts	Pediatrics
Hans Olsen	University of Virginia	Orthopedic Surgery
Joseph Ordonez	Beth Israel Medical Center / Albert Einstein	Family Practice
David Otto	St. Vincents Hospital / NYU Medical College	Med/Peds
Reshma Paranjpe	Mt. Auburn Hospital / Harvard Medical School	Medicine-Prelim
	Univ. Health Center of Pittsburgh / Univ. of Pittsburgh	Ophthalmology
Mark Payson	Walter Reed Army Medical Center	Ob/Gyn
Quynh Pham	Duke University Medical Center	Ob/Gyn
Michele Pierre-Louis	Pennsylvania Hospital / University of Pennsylvania	Ob/Gyn
Michael Posencheg	Mt. Sinai Hospital / Mt. Sinai School of Medicine	Pediatrics-Primary
Pedro Ramirez	Univ. of Alabama Hospital-Birmingham	Surgery
Vandana Reddy	Johns Hopkins Hospital / Johns Hopkins Medical School	Medicine
Silvana Ribaudo	NYU Downtown Hospital / NYU	Ob/Gyn
Eowyn Rieke	Swedish Medical Center / University of Washington	Family Practice
Jack Rozel	University Health Center-Pittsburgh / Univ. of Pittsburgh	Psychiatry
Susan Sabatino	Beth Israel Deaconess Medical Center / Harvard Medical School	Medicine
Pooja Sarin	Boston Combined (Children's Hospital) / Harvard Med School	Pediatrics-Primary
Andrew Shen	Beth Israel Deaconess Medical Center / Harvard Medical School	Medicine-Prelim
	UCLA Medical Center	Emergency Med
Neetha Shetty	Brown University Internal Medicine Residency	Medicine-Prelim
	Rhode Island Hospital / Brown University	Neurology
Julie Shih	NYU Medical Center	Ob/Gyn
Steven Shin	NYU Medical Center/Hospital for Joint Diseases	Orthopedic Surgery
Rakesh Singh	New York Methodist Hospital	Emergency Medicine
Jonathan Smith	Mt. Auburn Hospital / Harvard Medical School	Medicine
Jennifer Souther	Memorial Hospital / Brown University	Family Practice
Paul Spiegel	Beth Israel Medical Center / Albert Einstein College of Medicine	Medicine
Parvez Sultan	Univ. of Texas Medical School-Houston	Surgery
John Um	UMDNJ-RWJohnson-Piscataway	Surgery
Susan Walker	Cambridge Hospital / Harvard Medical School	Transitional/Psych
	Cambridge Hospital / Harvard Medical School	Psychiatry
Ron Willy	San Diego Naval Medical Center	Transitional
Albert Woo	Rhode Island Hospital / Brown University	Plastic Surgery
Yuki Young	Univ. of Michigan Hospitals	Medicine
Michael Zahalsky	Beth Israel Medical Center / Albert Einstein College of Medicine	Surgery-Prelim
	Beth Israel Medical Center / Albert Einstein College of Medicine	Urology
Dana Zappetti	New York Presbyterian Hospital / Cornell University	Medicine

* Two names withheld by request, and 4 students delayed match.

Table 4. State in Which the First Year of Residency Training is Located for the Brown University School of Medicine MD Class of 1999

Alabama	1	1.2%
Arkansas	1	1.2%
California	8	10.0%
Georgia	2	2.5%
Illinois	1	1.2%
Maryland	2	2.5%
Massachusetts	15	18.5%
Michigan	1	1.2%
Minnesota	1	1.2%
New Jersey	2	2.5%
New York	18	22.2%
North Carolina	2	2.5%
Ohio	3	3.7%
Oregon	1	1.2%
Pennsylvania	4	5.0%
Rhode Island	12	14.8%
Texas	3	3.7%
Vermont	1	1.2%
Virginia	1	1.2%
Washington	2	2.5%
TOTAL	**81**	**100%**

who enter residency programs in internal medicine end up in a subspecialty of internal medicine, such as cardiology. Likewise, about a quarter of those entering residency training in pediatrics will choose a subspecialty.

The data for Brown graduates are similar to the national AAMC longitudinal data. Of those Brown graduates in the MD classes of 1987-1991, only 46% of those entering residencies in internal medicine went into general internal medicine practice. In pediatrics, the figure is 59%. Among the primary care residencies, only family practice does not exhibit this pattern. Actually, the number of graduates in those five classes who eventually enter family practice is greater than those who started out that way by about 14%. The AAMC data are not very informative about subspecialization in obstetrics and gynecology. The Brown data show that 96% stay in the field of obstetrics and gynecology, but the proportion who subspecialize is not known. Applying this data to this class, about 33 graduates (39%) will actually practice in primary care.

The proportion of U.S. medical school graduates entering primary care specialties remained steady. For the fifth consecutive year, more than 50% of U.S. medical school seniors will pursue training in internal medicine, pediatrics, and family practice, according to the AAMC.[1] (The AAMC does not include obstetrics and gynecology among the primary care specialties.)

Table 3 lists the Class of 1999 graduates and their residencies. Of the 81 graduates who will enter residency training next year (4 are delaying their residencies for 1 or more years), 10 graduates (12%) matched with Brown-affiliated residency programs and will be staying in the state. New York City was the most popular metropolitan area for the graduates with 16 members of the class headed there. Boston was the second most popular locale, with 12 graduates locating there.

New York State will be the home for 18 graduates next year and ranks second to Rhode Island as the most popular state for residency training. (Table 4) A majority of the Class of 1999, 57%, will stay in the cold Northeast, up slightly from 55% in the previous class. The West Coast is a lower-than-usual choice of location with only 11% of graduates locating there, unlike the 21% of last year.

The drop in the proportion of graduates entering residency programs in primary care needs close monitoring in future years to determine whether this is a one-year aberration or a trend.

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Is It Time to Close the Book on Closed-Book Examinations?

Stephen R. Smith, MD, MPH

Abbreviations Used:

MCQ	multiple-choice-question
OSCE	Objective Structure Clinical Examination
PLME	Program in Liberal Medical Education
SAT	Scholastic Achievement Test
USMLE	United States Medical Licensing Examination

Every person reading this article has lived through it - entering the classroom with a supply of #2 pencils clutched in sweaty hands, struggling to take one last look at your notes before being ordered to close your book, clear your desk, and not open the exam book until told to do so, and then hearing "begin!" - and racing to get all the bubbles filled within the lines before hearing the dreaded, "stop!"

We've all done it before and will do it again, though perhaps the next time the #2 pencils will be replaced by a keyboard or mouse and the glossy exam booklet with a high-resolution computer screen. Though we've all learned to love to hate the closed-book, time-limited written exam, how many of us have questioned the wisdom of continuing this time-honored tradition?

My goal is to stimulate you, the reader, to question the rationale for continuing this tradition and to consider other ways to logically connect the method of examination to the goals of teaching. I will do so by briefly reviewing the minimal empirical research on the subject, touching on the theories of great thinkers, presenting a rationale for change, examining the barriers to change, and concluding with a strategy for change.

EMPIRICAL RESEARCH

I found very few articles in either the medical education literature or the general education literature on closed-book versus open-book examinations.[1-4] The research shows that open-book exams reduce students' perception of stress and anxiety, but performance differences are mixed with an overall net effect that seems not significant.

Students spend more time preparing for closed-book examinations, but spend more time taking open-book and take-home exams. The authors tend to support open-book and take-home exams as being more "satisfying" and having the potential to emphasize higher-order learning.

I did not find the articles very instructive. The authors did not seem to exploit the possibilities inherent in alternative assessment methods. Though they talked about the potential to assess higher-order learning, the tests they employed focused almost exclusively on measuring knowledge. Yet the literature and personal experience tell us that measures of knowledge are poor predictors of future performance as physicians.[5,6] In an article comparing medical school graduates who perform well in residency to those who perform poorly, Brown, Rosinksi, and Altman noted that the problems that beset the poorly performing residents were largely personal and motivational rather than knowledge- or skills-related.[7]

EDUCATIONAL PHILOSOPHY

The traditional fact-oriented, closed-book examination was criticized earlier this century in the final report of the Commission on Medical Education in 1932. "The present system of detailed subject examinations, which rely so largely upon memory and which are still popular in secondary schools and some colleges, tends to defeat the major purposes of the training, which are not the collection of facts but the intelligent and discriminating use of knowledge which is applicable to a given problem."[8]

George Miller laid out the case for different types of assessment in *Teaching and Learning in Medical School,* in 1961. "If a faculty believes specific facts are of primary or major importance, then facts should be taught and tested for; if the utilization of facts and skills in the solution of problems is regarded as the major goal, then this should receive major attention in instruction and appraisal."[9]

Donald Schön offers perhaps the most profound discussion of teaching, learning and assessment in professional education. He argues persuasively that the professional student learns by doing and proper assessment can occur only in the context of action. He describes the repartee between master and pupil in the act of doing, whether it is the mentor psychiatrist supervising a psychiatric resident stuck on a difficult case or the master architect instructing the graduate student struggling with the design of a school in topographically challenging terrain. Learning-in-action, teaching-in-action, and assessment-in-action become inextricably intertwined. Together teacher and learner struggle with the problem, the novice learning how to think like a psychiatrist (or architect, or musician, or whatever), becoming the professional step by step, with evaluation being the means by which the master guides and coaches the novice.[10]

RATIONALE FOR CHANGE

For the most part, we do not practice medicine the way we are tested in medical school. Except in emergency situations, physicians may consult a wide variety of sources as they attempt to make sense of their patients' problems. Indeed, such information-seeking behavior is praised and encouraged. Closed-book exams convey the message that the only right way is from memory; referring to other sources is, in a way, cheating. The ultimate purpose of evaluation is to measure how well the examinee will perform as a physician. The less authentic the evaluation, the less confidence we should place in the validity of the evaluation method. When the evaluation instrument requires that the examinee perform in a manner contrary to the way we wish him or her to perform as a physician, wary, indeed, should we be of that form of testing.

Evaluation drives the curriculum. Students will do whatever they need to do to pass a course. If the evaluation method used requires them to memorize vast quantities of facts, then students will do that. The hours needed to accomplish that task need to come from somewhere, whether it be from patient care responsibilities, personal time, or sleep. Before the clerkship in medicine adopted the Objective Structure Clinical Examination (OSCE) as a major component of the students' overall assessment, faculty were distressed to see the students all but disappear during the final weeks of the clerkship. They were in the library, or some out-of-the-way nook of the hospital or home, memorizing long lists of differential diagnoses, which they had heard was a favorite question of oral examiners. Once the OSCE was in place and the oral exam eliminated, student behavior changed radically. Instead of disappearing, students were everywhere - asking residents to show them again how to palpate the spleen, in the radiology suite brushing up on chest x-ray interpretation, even in the lab asking technicians to help them read Gram stains! This pleased the faculty immensely.

This intensive learning behavior on the part of the students was high in energy, but not nearly as anxiety-provoking as the old *viva voce*. Students consistently describe the OSCE on the clerkships as engendering a healthy amount of anxiety and actually being fun to do and instructive to boot.

... the literature and personal experience tell us that measures of knowledge are poor predictors of future performance as physicians

◆

Beyond the diminished stress and anxiety, performance-based assessment reduces the "we-versus-them" attitude in which students begin to believe that the faculty are "out to get them." Typically, in the closed-book examination situation, students worry that faculty may spring something unexpected on them on the test. Faculty, on the other hand, complain that students are conniving to find out what will be on the test. Faculty fear that if they tell the students what will be on the exam, the students will limit their studying to those topics rather than seek a broad-based understanding. In contrast, performance-based examinations are characterized by explicit, publicly shared expectations. Rather than trying to trick students, faculty hope that all students will perform well. In the obstetrics and gynecology clerkship OSCE, for example, the clerkship director tells the students what each of the OSCE stations are at the beginning of the clerkship. S/He wants every student to be able to teach breast self-examination, to perform a skillful pelvic examination, to demonstrate the hand maneuvers for a normal delivery, etc. Students view him as an ally in their learning, not an enemy.

These few examples illustrate the tight connection between the teaching goals and the assessment methods. If one goal of the ob/gyn clerkship is to have students learn how to teach breast self-examination, then the best way to figure out whether they mastered that goal is to watch them teach a patient (or, in this case, a standardized patient) breast self-examination. This concept is not confined to clinical clerkships. In anatomy, students are expected to relate anatomy to clinical medicine. They are assessed by presenting a demonstration of the area under dissection to the faculty. In my own course on health policy, students select a topic, research it, analyze it, and write a paper on it. One student a few years ago criticized me for not giving a closed-book exam on the 1365-page Clinton Health Securities Act. What a waste of the students' time it would have been to memorize the details of that mammoth piece of legislation, which would shortly thereafter end up in the dustbin of history!

While I had read every page of that bill, I would never trust my own memory about specific facts. Closed-book examinations ignore modern technology for rapid information retrieval. The Internet offers physicians access to information in ways never imaginable just a few years ago. Office-based computer systems can give physicians differential diagnoses, practice guidelines, drug interactions, referral sources, and more at the touch of a key. Testing students on their ability to match memory capacity with computers is an anachronism. Instead of forbidding students to bring "extraneous materials" with them into the examination lecture, we ought to be requiring them to bring their computers and prove to us they know how to use them to get needed information.

Wise words from a distinguished member of the United States Supreme Court highlight the folly of memorizing facts. Mr. Justice Hugo Black carried a copy of the Constitution in his pocket. A reporter asked, "Why do you carry that? By now, you must know it by heart." Mr. Justice Black replied, "Indeed, I do not, and each time I read it, I find something new in it."[11]

Lifelong learning gets a lot of lip service in medical education, but, as George Miller said, "an objective is real only when a faculty attempts to do something about it"[12] Cramming for closed-book exams does not promote lifelong learning. The rest of life is an open-book exam that tests character as much as intellect. Again, to quote from the late George Miller, "true learning begins with not knowing the answer."[13] In a recent visit to a medical school in a developing country, I sat in on two basic science classes. Throughout the lecture, students would call out the answer to the oral "fill-in-the-blank" cadence of the instructor. The students were sharp; they knew the right answers. After class, however, I was mobbed by students who complained of the long hours they had to devote to studying for the detailed, fact-oriented exams. Students spoke of studying nine hours a day outside of class (substantiated by the faculty). Later the same day I met with students in their final year of medical school. One of their biggest complaints was the conflicting positions taken by their faculty on clinical problems. They were uncomfortable not having a single "correct" answer; they wanted to be told the right way. Though I may be erroneously drawing conclusions from this "quick-and-dirty" cross-sectional study, I viewed the concerns of the sixth-year students as the logical outgrowth of the way the second-year students were being taught and evaluated. The students were enmeshed in a system that stressed "the right answer" rather than one that urged students to ask the right question. Not enough emphasis was placed on dealing with conflicting "truths," of evaluating the evidence, or, as Thomas Jefferson urged the medical students at the University of Virginia, "to maintain a wise infidelity against the authority of his instructors and the bewitching delusions of their theories."[14] The curriculum - specifically, the methods of evaluation - was not fostering skills in lifelong learning, despite the professed desire of the faculty to do so. Much to its credit, this foreign medical school recognized this and was undertaking a major curriculum reform.

BARRIERS TO CHANGE

Closed-book, time-limited examinations remain the dominant form of assessment in medical schools across the United States and throughout the world, despite all the reasons to change enumerated above. Why?

External standardized certifying and licensing examinations represent the single greatest barrier to change. Clones of these machine-scored, multiple-choice-question (MCQ) examinations are administered by faculty throughout the medical curriculum, both at the undergraduate and residency levels, in the belief that practice makes perfect. This is a dubious assumption. The strongest predictor of performance on an MCQ exam is performance on a previous MCQ exam. The correlation between the verbal Scholastic Achievement Test (SAT) score and Step 1 of the United States Medical Licensing Examination (USMLE) was 0.350 among 330 Brown students in the Program in Liberal Medical Education (PLME), and 0.319 for Step 2. The correlation between Step 1 and Step 2 was 0.738. A good likelihood exists that strong performers on MCQ tests will continue to perform well whether or not they had the opportunity to practice taking similar exams dozens of time before. This hypothesis has not been tested, however, and medical schools seem reluctant to be the first to challenge that assumption.

The heavy reliance on closed-book, time-limited MCQ exams might be justified if they were robust predictors of future performance of the examinees as physicians. Yet these exams are woefully inadequate in this regard. The correlation between the SAT verbal score and ratings by residency program directors of Brown PLME graduates was actually negative, at -0.055! Similarly, the correlation between national board scores and residency program directors' ratings of Brown graduates was also weak, ranging from 0.065 to 0.172.

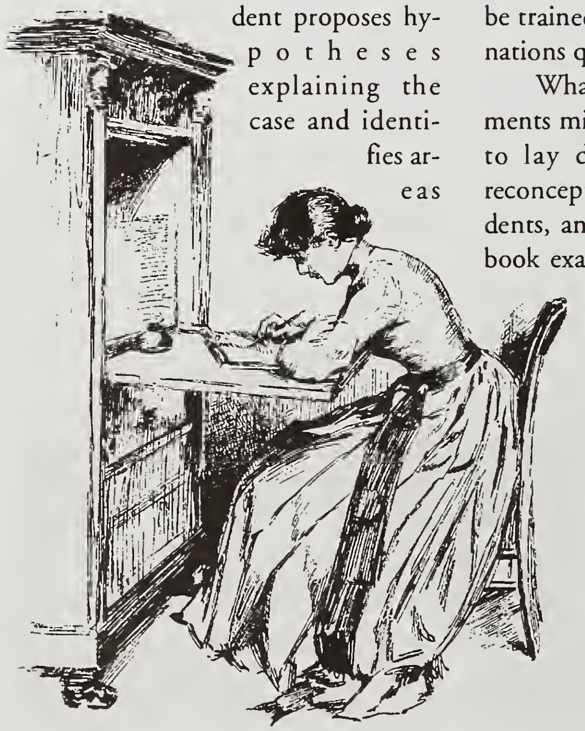
The ease of administering a machine-scored examination simultaneously to thousands of candidates has been the primary factor behind the choice of this type of examination by licensing and certifying bodies. Yet in their defense, these examinations are not meant to be considered in isolation. The National Board of Medical Examiners contends that its exam only tests a small proportion of the competencies needed to practice medicine. Medical schools and residency programs are supposed to be assessing the other competencies. Licensing and specialty certification require not only a passing score on an MCQ exam, but also the endorsement of the faculty, represented by the doctor of medicine degree and certification of successful completion of residency training. It would make sense for medical schools to use methods of assessment different from the MCQ exam if the licensing authorities have assumed the responsibility for assessing knowledge through the use of an MCQ exam. Such a multimodel approach to assessment is what is envisioned by the combination of a licensing exam and the awarding of a degree.

The same ease of administration appeals to faculty in their own courses. The familiarity of the closed-book examination makes it the first consideration when faculty choose a method of assessment. Alternate forms of assessment are likely to be less familiar to faculty, who would be concerned about the credibility with their colleagues of nontraditional approaches to assessment. They would also be likely to be less confident in grading student performance or designing the assessment instruments.

STRATEGIES FOR CHANGE

I propose that teachers in every course design two kinds of assessments. The first - a typical closed-book examination - would assess the students' ability to commit to memory those facts that every physician should know immediately and without need to refer to outside sources. These are facts that would be known accurately by 90 out of 100 practicing physicians "off the top of their heads." The teacher would prepare a list of these facts and tell students that the examination would sample from this list and only from this list. Students would be expected to get 90% of the answers correct or repeat the exam. Students could not pass the course until they achieved 90% accuracy or better. These exams could take no longer than half an hour and be given no more often than every four weeks during the semester.

The second type of assessment would expect students to function like physicians, namely, with full access to all outside resources and ample time to explore their questions. This could take the form of the familiar take-home examination, or a research paper, or more innovative assessment techniques, such as the "triple jump" pioneered at McMaster University. In the triple jump, the student is presented a case by the examiner. In the first "jump," the student proposes hypotheses explaining the case and identifies areas where further information is needed. The second "jump" involves the student's pursuit of the additional information, usually for several hours. The student returns to the examiner for the third "jump," at which time the student describes how the additional information was sought, what was found, and how that information changed or supported the original hypotheses. This technique allows the examiner to assess the student's problem solving, information retrieval skills, critical thinking, understanding of the content area, and communication skills.



In the human morphology course at Brown, students in groups make oral presentations to the faculty, focusing on the area of dissection for that unit of the course. Faculty assess the students on their knowledge of anatomy and their ability to apply the basic science of anatomy to clinical medicine, to exhibit the skills of a lifelong learner, and to communicate clearly. This has proven to be a highly regarded approach to assessment by students and faculty alike.[15]

These alternative assessment techniques require more time for the faculty to grade, though they take less time to design than MCQs. The task can be divided among several faculty to make it less burdensome. Graduate and medical student teaching assistants can be trained to grade take-home examinations quite reliably.

Whatever the additional requirements might be, we must be prepared to lay down our No. 2 pencils, reconceptualize the ways we assess students, and close the book on closed-book examinations. The information revolution that illuminates the dawn of the new millennium demands no less.

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To Dream, Perchance to Sleep

Garey Noritz, MD

Commencement Address, Brown University School of Medicine – May 31, 1999

"For this is the day of joy
which has been fourteen hundred and sixty days in coming
and fourteen hundred and fifty-nine nights."
—John Stone, MD

Today is a day to rejoice.
A day to accept congratulations.
To hold our heads up high for all we have learned.
To try and hide for all we have not.
To prepare for responsibilities too numerous to count.
As we swear like sailors as our pagers pipe us to duty.

Today is a day for us,
As our other days will be in the service of others.

Today we stand with our patients through their lives,
And to their deaths
So that no one makes the journey alone
Or afraid.

Today we walk to the delivery room,
We carry to the nursery.
We give the shots and chart the growth.
Through cold and flu, and broken legs,
We hold the hand from ER to OR,
So that Grandma may hold hands again.

Today we become teachers,
Of patients and students,
And ourselves.
Of our friends and family, who can, by now, tie the knots and name the cranial nerves,
Having stood by us these fourteen hundred and sixty days.

Today our successes will be triumphant, often unthanked.
And thanked generously, when all we have done is let nature take her course.
We fear, every moment, our failures, which haunt us at night.

Today we try to remember the millions that we have forgotten:
Which is Berger's Disease?
And Which Buerger's?
A menagerie of tests: a CAT scan; a PET scan;
Haunted while we eat by CABGs and lumbar lams.
The HELLP syndrome.
VATER, who is not Luke's father.
We fear ARDS.
We have learned another language.
We guide and translate in a strange land.

Today we look forward to another's illness.
We fear, deep down, that we will be somebody's "great case".
To treat each patient as a human being,
When we are zombies ourselves.

Today we scold for high cholesterol
While we have a belly full of ribs.

Today we give solace when our own hearts are breaking.

Today we resist temptation
As we remember that we are human,
As it is expected that we are not.
Our frailties in sharp focus beyond our bleary eyes.

Today we count the triumphs,
As there will be many to come.
Infections cured,
Pain alleviated,
Bad habits broken,
Stones removed,
Demons chased.

Today we wonder forever what else to do.
We know what else to do, but know it should not be done.

Today we narrate during ER and MASH.
We focus on plot instead of character.

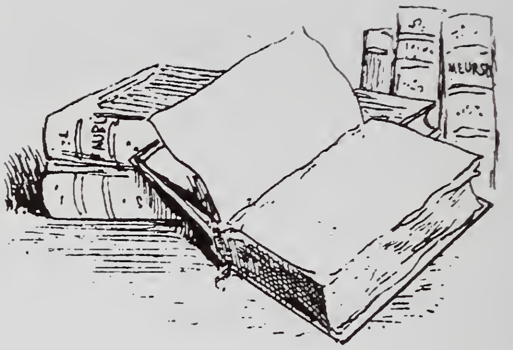
Today we give comfort, though not reimbursable.
It is how we earn our keep.
And pay back those who put us here,
Sweating beneath our robes.

Today we mediate
Between mother and fetus,
And mother and father,
And father and son,
Between physician and surgeon,
And Blue Cross and Blue Shield.
Between heart and kidney.
Between the rock and the hard place.
Our id and our ego.
And ourselves and our loved ones.

For none of us is an island,
Though that's what we see
when we close our eyes and dream.
Grass skirts and passionfruit
Instead of johnnies and normal saline.
To see the sun, not through a window,
But beating down upon us.
To feel the sand beneath our feet,
Oh, To dream, perchance to sleep.

Today we know the whats and the hows but not the whys.
For the whys are the art
And the science is always changing.
Though the art will serve us forever.

For Hippocrates had his humors,
Hahnemman his homeopathy,
Mesmer his magnetism,
Today we have our "evidence" to guide us.
Tomorrow, new textbooks will call us primitive,
Though the art will serve us forever.



Today we try and live up to the hopes of our friends and families, our mentors.
And most of all, ourselves and our patients,
We love the art but live on the science.
We know both must be taken together,
But only realize it when one is missing.

Today we sit together, the twenty-fifth class.
We remember the trying times, the dark times, the triumphs.
The sweaty palms of anatomy,
Bloodshot eyes peering into microscopes, sorting pink from purple
The stiff necks studying for the boards,
The shaking hands, drawing blood for the first time.
The screams and shouts of match day,
And now the applause as we are delivered by Providence.

Today is a day to be proud.
To be scared.
To be in love.
To celebrate as only brand new MDs know how:
To stand and cheer, and take a bow.

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Charles Whitney Page, MD, and the Abolition of Restraints in American Psychiatry

Lyman A. Page, MD

“Since that time I have been responsible for the custody and treatment of more than six thousand insane persons, not one of whom was restrained with mechanical appliances. . .” [1] “That time” was 1897, when Dr. Charles Whitney Page finally succeeded in eliminating restraints completely from Danvers State Hospital in Danvers, Massachusetts. His article recounts the history of this reform, beginning with Philippe Pinel during the French Revolution. Pinel was shocked at the chaining of “maniacs” in Bicetre, “the great Paris asylum for incurable insane men,” removed the chains, and encountered no untoward events. This result was not widely acknowledged, and when the Quaker, William Tuke, established at his own expense at York, England, a hospital in which “lunatics were treated as sick ... and gentleness and patience would ... be exercised toward them,” he encountered tremendous hostility and vilification, “especially [from] those interested in the management of English asylums.”[1] In 1838 at Lincoln Asylum Drs. Gardner Hill and Charles Worth were fired for trying to abolish restraints. But in 1839, by dint of tremendous effort and attention to details, Dr. John Conolly ended restraints successfully at Hanwell Lunatic Asylum, which held over 800 patients, and began the slow process, against many detractors, of showing by example that continued use of restraints was “irresponsible within the limits of civilization.”[1]

Charles Whitney Page, MD, was one of the pioneers of the modern era of treatment of the “insane” in America. He was born in Westminster, Massachusetts, on September 7, 1845, attended Dartmouth College and Harvard Medical School, and worked as a general practitioner until 1874, when he became resident physician in psychiatry at the Hartford Retreat. He remained on the staff there until 1888, when he became Superintendent of the Danvers State Hospital. He resigned his Danvers position in 1898 to become Superintendent of the Connecticut State Hospital for the Insane at Middletown, where he instituted, as he had at Danvers, common dining for the inmates. (In 1955 the Congregate Dining Hall was named Page Hall and with recent renovations has become the central building in the Hospital.[2]) In 1901 he resigned from the Middletown position (becoming the shortest-tenured Superintendent in the Hospital’s history[2]) to reassume (at higher pay[3]) the post at Danvers. He remained there until 1910. During his time at Danvers he served as president of the New England Society of Psychiatry. On retiring from his Danvers position he returned to Hartford and took up residence in a house his second wife inherited from her aunts. He soon became a Board member of the Good Will Club in Hartford, and was its president for 15 years, retiring in 1931. That organization provided educational and recreational facilities for underprivileged boys in the Hartford area. He died in his sleep of “bronchitis and a weak heart” in the early morning of February 16th, 1932.[4] He had been working on a monograph about a colleague until the week he died.

> *Dr. Page’s abolition of physical restraint of the “insane,” his socialization of the inmates in the institutions he directed, and his perception of the potential importance of laboratory investigation of psychiatric patients were early changes in psychiatry in America presaging the modernization of treatment and attitudes towards psychiatric patients in a process that is still continuing...*

While at Danvers, Dr. Page oversaw major changes in the facility in addition to the reforms on the use of restraints and eating arrangements.[5] He battled constantly with the State Legislature for more facilities to care for an ever-expanding patient load. He met the problem of inadequate nursing by establishing a nursing school in the facility, which grew erratically until his retirement and provided some fine Hospital staff. This school was said to be the first of its kind in this country to be established in connection with a public institution.[5] He was a pioneer in advocacy and funding of adequate clinical laboratory facilities in hospitals for the insane, and from the mid-1890s the Hospital staff published several papers annually on pathological and chemical analyses on their patients. Dr. Page emphasized that, particularly with their proximity to Harvard, there were different missions of clinical and research laboratories, and that their work should be complementary and cooperative, not competitive. His interest and pride in the growth of the laboratory investigations (and publications) at Danvers and the increasing numbers of autopsies through the years reveal an implicit psychobiological orientation, although he never explicitly discusses this.

Abolition of the use of restraints was not an easy process.[5] Dr. Page's first few annual reports refer to use of restraints reluctantly and report the numbers of instances almost guiltily. By 1892 he could say "our experience . . . demonstrates that mechanical restraints can be reduced to infrequent application, if not entirely abolished, in lunatic hospitals, when the officers desire the success of the non-restraint plan, and especially when attendants become interested or enthusiastic. . ."[5] Restraints were finally abolished on the women's ward by 1893, 3 1/2 years before their abolition on the men's ward, because of greater willingness of the attendants to do so. In 1896 only one suicidal patient was restrained mechanically, and in 1897 Dr. Page reports the complete avoidance of mechanical restraint. It was in that year that the "conjugate dining room" was opened and became a permanent fixture, with its platform on which the orchestra played during every meal, 1 hour for dinner, and 30-40 minutes during breakfast and supper. Dr. Page could not understand why the use of music had been condemned elsewhere and surmised that it related to the competence of the musicians: "good music is important." It was about this time that attendants' uniforms were abandoned, as an additional measure to make patients feel a part of the community.

There were nonbelievers. In his 1897 report[5] Dr. Page wrote "It is often asserted that chemical restraint . . must be used . . . where mechanical restraint is abolished. Such is not the fact." He then lists all the drugs dispensed to the 857 patients through the year: 58 doses of sulphonal, 1 dose of chloral, 2 "cerebral sedatives," 16 doses of liq. opii. comp., 12 doses (1 grain each) of codine [sic],2 doses each of 1/100 grain and 1/80 grain hyoscine, 150 grains of K/Br daily to 5 epileptic patients, opium nightly (1/2 grain) for pain to one woman, 1 dose of morphine for acute pain, and Squibb's mixture and tinct. opii. camph. for "bowel trouble." Two admitted addicts received gradually decreasing opiate doses for 15 and 18 days respectively.

WALPOL
VOLTAIRE
BURKE
COMPROMISE
MISCELL
RESIGNATION

Hydrotherapy, which was very popular,[6] was employed on some patients, but Dr. Page took pains to point out that it was not used as restraint, since popular methods involved very restraining techniques in which patients were tightly wrapped for hours at a time. At Danvers no dry packs were used and the cold, wet bandages were applied to the limbs separately. This therapy was used for "prolonged melancholia," but the Danvers experience was too limited to warrant conclusions, and Dr. Page referred readers to the larger experiences of the Vanderbilt Clinic in New York City and the German Hospital in Philadelphia.

Likewise, seclusion was severely cut back, and by the late 1890s was used only to protect other patients, and then for brief periods. Page came to believe that shorter periods (hours) of seclusion were not only more humane, but more effective than longer periods, and he used it decreasingly, and almost always for safety, "because of the crowded condition of the wards."[5] From his earliest days he would startle (and educate) the staff by entering alone the rooms of patients considered dangerous, and he once got accidentally locked into such a room, so that the security staff had to break down the door. He emerged unharmed.[7]

Page's emphasis on the beneficial effects on caretakers of the abolition of restraints for patients is noteworthy. For example, his 1897 report states "As long as nurses understood that straps and jackets could be employed as final measures, they not only relinquished mild efforts too quickly, but were inclined to assume a dictatorial, aggressive manner towards patients . . . Certainly the non-restraint rule has established a kindly, humane spirit in our wards as no other measure could have done."[5] This is reminiscent of Harriet Beecher Stowe's repeated insistence that one of the vilest results of the institution of slavery was the brutalizing effect it had on the slave-holder.[8]

Dr. Page's abolition of physical restraint of the "insane," his socialization of the inmates in the institutions he directed, and his perception of the potential importance of laboratory investigation of psychiatric patients were early changes in psychiatry in America presaging the modernization of treatment and attitudes towards psychiatric patients in a process that is still continuing, a process that supports Bech's contention that "Man is in the awkward position of being kinder than Nature."[9]

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Bungee Jumper's Eye

Rochelle Chodock, BA, Philip Fisher, MD, Mark Rosenthal, MD, Daniel Kletzing, MD, FACS, Patrick J. Maloney, Marzouq A. Qubti, Mark Walsh, MD, FACEP

Para-nasal sinus barotrauma and resulting infection associated with bungee jumping have not been reported in the medical literature. We report a case of periorbital cellulitis which developed after bungee jumping in a thirty year-old man with previously treated sinusitis. Rapid changes in barometric pressure have been known to cause "aviation sinusitis;" and in analogy with this condition, we postulate that the rapid deceleration of the head as the bungee reaches its limit and begins to spring back increases pressure on the lining of the para-nasal sinus. This allows the translocation of bacteria from infected para-nasal sinuses to the periorbital space. Since many people who have sinusitis may participate in bungee jumping, it would be reasonable to assume that more cases of extension into the periorbital area may present following participation in this activity.

> *We report a case of periorbital cellulitis precipitated by bungee jumping.*

INTRODUCTION

Bungee jumping can be defined as the act of jumping while attached to an elastic cord from a high altitude. The jumper rebounds after the cord reaches maximum length falling again once or twice before being either lowered to the ground or pulled up to the point of the initial jump. Most jumpers are attached to the cord by a vest. In the past the cords were attached to the ankles, however, the vest is becoming the predominant method of attachment of cord to the jumper. Fatalities and injuries resulting from bungee cord jumping have been described in the medical literature. These injuries have been the result of a failure of the cord to remain fastened to the jumper, an improperly long cord, the collapse of the supporting structure, or knotting of the cord in a fashion so as to impinge neurologic or musculoskeletal structures.[1,2] However, there are no reports describing periorbital infection following bungee jumping. We report a case of periorbital cellulitis precipitated by bungee jumping.

CASE HISTORY

A 30 year old man presented to the emergency department with painful swelling of the left eye. Two weeks earlier he had completed a ten-day course of amoxicillin for left-sided para-nasal sinusitis. The patient's symptoms of purulent nasal discharge, fever, and left-sided facial pain improved, but the left frontal headache persisted. Three days prior to admission, the patient went bungee jumping. Minutes after jumping, the patient noted pain behind the left eye and worsening of the left frontal headache. The patient had a history of one previous episode of uncomplicated sinusitis. Over the next three days, progressive swelling and redness of the left eye developed along with 101.1°F oral temperature. Evaluation in the emergency room revealed significant left palpebral erythema and edema with reduction of the palpebral fissure by 50%. Left conjunctival erythema and edema were present. Extraocular motion was full yet painful in the left eye. Slit lamp revealed a normal cornea and anterior chamber. Fundoscopic exam showed a normal left retina. The remainder of the physical examination was unremarkable. A complete blood count was normal.

Routine sinus films revealed hypoaeration of the left frontal sinus consistent with sinusitis. The CT scan of para-nasal sinuses revealed asymmetric soft tissue thickening in the left pre-septal orbital region consistent with periorbital cellulitis.

A diagnosis of left frontal sinusitis with extension to a left periorbital cellulitis was made and the patient was admitted to the hospital. He was given one gram intravenously of aztreonam every eight hours and one gram of nafcillin every four hours. Cultures of the minimal exudate from the left eye grew mixed gram positive flora. By the third day of hospitalization his periorbital pain and swelling subsided and he was afebrile.

DISCUSSION

Since the first commercially sponsored jump in 1988 there have been more than one million bungee jumps. Multiple fatalities have been reported.[2] Nonfatal injuries include paralysis due to spinal cord injury, peroneal nerve palsy; cerebral concussion; various musculoskeletal injuries including gluteal hematoma, sprained fingers, abrasions, bruises; and cord burns on hands.[2] Visual loss and subconjunctival chemosis and hemorrhage have also been described.[3,4]

This represents the first reported case of bungee jumping induced periorbital cellulitis. That such a complication should follow bungee jumping in a patient with pre-existing sinusitis is not surprising. The close proximity of the orbital structures to the para-nasal sinuses frequently leads to the extension of infections from the sinuses to the neighboring orbit. Anatomically, the maxillary, frontal, ethmoid, and sphenoid sinuses comprise the para-nasal sinuses. These four sinuses surround the nasal cavity, communicating with the nasal cavity by means of natural ostia. The ethmoidal sinus is separated from the orbit by the very thin and marrowless lamina papyracea. The floor of the frontal sinus contributes to the roof of the orbit and the roof of the maxillary sinus contributes to the floor of the orbit.[5]

The majority of infections occur in those sinuses that open into the middle meatus. Those sinuses are the maxillary, the anterior ethmoid, and the frontal sinuses.[5] Complications of sinusitis can be defined as local, orbital, or intracranial. The most common of these complications is the extension of the infection from the sinus into the orbit. Often, the infection spreads directly from the ethmoid sinus through the thin lamina papyracea into the orbit. This thin bone represents a poor barrier to bacterial spread. Likewise, a frontal sinusitis can spread to the orbit because the floor of the sinus forms part of the roof of the orbit. Once infection spreads into the orbit, inflammation and swelling of the eyelids will develop. In orbital cellulitis there is a diffuse edema of the orbital contents and infiltration of the adipose tissue with inflammatory tissue. Proptosis, chemosis, and decreased visual acuity will follow. With time a subperiosteal abscess, defined as a collection of pus between the bony wall of the orbit and the periorbital tissue, may develop and present as increasing proptosis with displacement of the globe laterally or downward. The orbital abscess is a discrete collection of pus in the orbital tissues and causes exophthalmus, chemosis, and complete ophthalmoplegia.[6]

Therefore, an inflammatory process can extend from any para-nasal sinus to the orbit through either the bony wall or the venous circulation.[6,7] The description of orbital cellulitis following a violent sneeze in a patient with chronic sinusitis suggests that barotrauma can facilitate the spread of infection from the para-nasal sinuses to the orbit.[8] After swimming or diving a patient may develop purulent nasal discharge and headaches followed by proptosis, chemosis and reduced visual acuity, indications of orbital cellulitis.[8] Rapid changes in barometric pressure can cause "aviation sinusitis."[9] By analogy with these circumstances we postulate that the rapid deceleration of the head as the bungee reaches its limit and begins to spring back can also cause barotrauma. An increase in pressure on the lining of an infected para-nasal sinus can thus result in periorbital cellulitis. We recommend that patients with true bacterial sinusitis complete the current recommendation of two week treatment with appropriate antibiotics prior to bungee jumping.[10]

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Clinical Performance Measurement

Edward Westrick, MD, MS

Clinical performance measurement is becoming an increasingly important issue in today's health care delivery system. In this month's column I will provide some historical context, a conceptual framework, and a report on progress at the state and national levels.

Clinical performance measurement can be used essentially for two purposes: quality improvement and accountability. Performance measurement for accountability purposes requires more rigorous measurement methodology because of the consequences of these uses. These can include public reporting and performance evaluation. These evaluations may compare the performance of specific organizations with each other. When performance measurement is used for quality improvement purposes comparisons are typically made within an organization over time. These measurements are used for managing processes and therefore don't need to be as methodologically rigorous. The information is typically used by organizations to identify opportunities for system improvement and to determine if system changes are accomplishing their desired goals. RIQP uses performance measurement exclusively in the quality improvement context.

The modern science of quality measurement can be traced to the work of a physician, Avedis Donabedian, who proposed a framework for this more than 30 years ago. His framework categorized measurement into structures, processes, and outcomes. Think of structures as the people, places, and things in health care. Think of processes as the verbs in health care, what the structures do. Outcomes are the results of structures doing processes. In health care quality measurement we look at indicators at all three levels.

Examples of structural measures include: number of hospital beds, staffing ratios, and proportion of board certified physicians on a panel. Early quality measurement relied almost exclusively on structural measures. Process measures are more commonly used these days. These include most of the indicators that RIQP works with such as: beta-blocker use after acute MI, ACE inhibitor use in CHF, dilated funduscopic examination in diabetes mellitus. Outcome measures are of greatest interest since the results are what we all want to know. Outcome measures can be divided into three categories: clinical, humanistic, and financial. Clinical outcome measures include mortality and morbidity end-points. The humanistic measures include patient satisfaction, functional status, and quality of life. Financial outcomes include both the costs of health care and the costs avoided by providing health care.

Abbreviations Used:

AAFP	American Academy of Family Physicians
AAHP	American Association of Health Plans
ACP	American College of Physicians
ADA	American Diabetes Association
AHCPR	Agency for Health Care Policy and Research
AHQA	American Health Quality Association
AMA	American Medical Association
AMAP	American Medical Accreditation Program
APhA	American Pharmaceutical Association
DQIP	Diabetes Quality Improvement Project
HARI	Hospital Association of Rhode Island
HCFA	Health Care Financing Administration
HEDIS	Health Plan Employer Data and Information Set
JCAHO	Joint Commission on the Accreditation of Healthcare Organizations
NCQA	National Committee for Quality Assurance
PMCC	Performance Measurement Coordinating Council
RIMS	Rhode Island Medical Society
RIQP	Rhode Island Quality Partners
SCRIPT	Study of Clinically Relevant Indicators of Pharmaceutical Therapy
VA	Veterans Administration

Intermediate outcomes can also be measured. These include blood pressures, LDL levels, and glycosylated hemoglobin levels in patient populations. Process measures and intermediate outcome measures are used more than distal outcome measures because they occur with sufficient frequency and speed to serve as performance measures. Distal outcome measures like mortality often take too long and are too infrequent to provide meaningful measurement.

In Rhode Island we are currently working with the Department of Health and other important stakeholders, including the RIMS, to develop a health care quality performance measurement and reporting program for licensed health care facilities. This effort was directed by state legislation passed approximately one year ago. The program will include measurement for both accountability and quality improvement purposes. Early work is focusing on hospitals and will be followed by long term care facilities and home care agencies. Health plans will not be included since they are covered in other legislation. Physician performance is not included in either sets of legislation. Since the early work is focused on hospitals and there are other reporting requirements for hospitals, both locally and nationally, a discussion has started among the HARI, JCAHO, RIQP, and HCFA to try to develop a set of core measures that satisfies all concerned so that the measurement burden is minimized. RI may become a model state for the rest of the country to follow.

There are primarily three private sector organizations that are leading performance measurement in health care today. The JCAHO primarily accredits hospitals and is a leader in hospital performance measurement. The NCQA accredits health plans. HEDIS is the performance measurement set that NCQA uses to evaluate MCOs. The AMA has recently entered this game with the American Medical Accreditation Program (AMAP). This program seeks to accredit physicians. These three organizations recently formed the Performance Measurement Coordinating Council (PMCC) with the goal of minimizing discordant measurement across settings of accreditation. Medical specialty boards are likely to develop physician performance measures in the near future.

There are two national efforts designed to accomplish this same goal. The Diabetes Quality Improvement Project (DQIP) brought together the important stakeholders in diabetes care to create a common set of measures to evaluate quality in the care of patients with diabetes mellitus. The DQIP measures have been adopted by the NCQA and the HCFA. In addition, the ACP, AAFP, VA, and ADA formed the core of DQIP. The SCRIPT Project is designed to develop a common set of measures to evaluate the quality of medication use. This effort will focus first on medication use in cardiovascular disease. The SCRIPT Project works under the auspices of a national coalition of 50 organizations who share this interest. Original members include: AMA, HCFA, JCAHO, AHQA, APhA, NCQA, AAHP, and AHCPR.

The efforts of these organizations use common criteria in varying forms to evaluate potential performance measures. I will share one version here. Performance measures should focus on clinical topics that are important and meaningful. There should be a relatively high disease burden (prevalence and severity), and potential for improvement. Measures should be based upon guidelines supported by scientific evidence and by expert consensus when sufficient scientific evidence is lacking. Measure components should be well-specified including procedures for data collection and analysis. Measures should be tested with scientific rigor to establish reliablity and validity. Measures should focus on clinical topics where there are opportunities to provide cost-effective care. The measurement process must be feasible, reducing data collection burden as much as possible. The measures should be interpretable by consumers, clinicians, and researchers.

This brief description on clinical performance measurement will be followed by a special issue of this journal on health care quality measurement after significant progress has been made on these local and national efforts. Look for this special issue next year.

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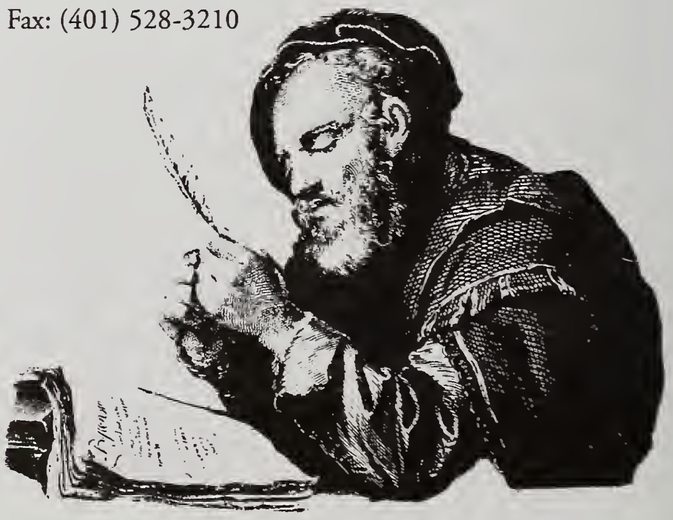

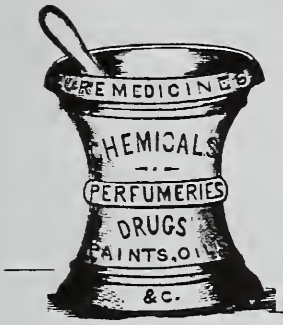

Advances in Pharmacology

Antiplatelet Agents and the Role of Clopidogrel in the Prevention of Secondary Atherosclerotic Events

Anita Jackson, PharmD, and Andrea F. Luisi, PharmD, BCPS

Cardiovascular disease is the leading cause of mortality in the United States, responsible for more than one million deaths annually.[1] The pathogenesis of atherosclerosis is a multi-factorial process involving endothelial damage, lipid accumulation, platelet aggregation, and plaque formation.[2] Platelets play a role in both plaque formation and the development of an acute thrombus following plaque rupture. This article will describe the process of platelet aggregation, review the pharmacology of the available antiplatelet agents, and discuss clopidogrel and its role in the secondary prevention of atherosclerotic events.

Activated platelets adhere to injured vessel walls and release agents that promote further adherence and aggregation. Following platelet activation, a variety of factors are released which transform the glycoprotein IIb and IIIa receptors into a complex known as GPIIb/IIIa. This complex binds to fibrinogen resulting in platelet aggregation.[3] The surface of the platelet aggregate allows the progression of the clotting cascade, with subsequent thrombin and fibrin formation and stabilization of the platelet plug.[1]

Several antiplatelet agents are available for primary and secondary prophylaxis of platelet-mediated atherosclerotic events. Aspirin preferentially inhibits cyclooxygenase-1 (COX-1) to decrease the production of thromboxane A2, a platelet aggregator. This inhibition of COX-1 is irreversible and, therefore, antiplatelet activity remains for the life of the platelet (approximately 10 days).[4] COX-1 is expressed in platelets, whereas COX-2 can be induced in response to inflammatory stimuli. Aspirin has been shown to be effective for secondary prophylaxis of cardiovascular disease including myocardial infarction and stroke.[5] Recommendations for patients with documented cardiovascular disease include the use of aspirin in doses of 160-325 mg daily.[6] Studies have also shown a benefit from aspirin for the primary prevention of myocardial infarction.[7,8] The aspirin dose recommended for primary prophylaxis ranges from 81-325 mg daily.

Abbreviations Used:	
MI	myocardial infarction

Dipyridamole is a pyrimidopyrimidine derivative with purported vasodilator and antiplatelet properties. The exact mechanism of dipyridamole's antiplatelet action is not known; however, two main theories exist. First, dipyridamole appears to be a phosphodiesterase inhibitor, which would prevent the complexation of GPIIb and IIIa receptors on the surface of the platelet. Dipyridamole may also stimulate prostacyclin synthesis, which has antiplatelet actions of its own. The clinical efficacy of dipyridamole has been questionable in the past. A recent trial has been published with positive results (the European Stroke Prevention Study-2). This study compared placebo, low-dose aspirin (50 mg per day), dipyridamole (400 mg per day), or aspirin plus dipyridamole in over 6000 patients with a history of stroke or TIA. All three treatments resulted in a significant decrease in the risk of a subsequent stroke. A high-dose, sustained release formulation of dipyridamole was used in this trial, which is not currently available in the United States. The combination of dipyridamole and aspirin used in this study showed the highest rate of stroke prevention.[4,9]

Glycoprotein IIb/IIIa receptor antagonists bind to the GPIIb/IIIa receptor, which prevents the receptor from complexing with fibrin. The agents currently available are abciximab, eptifibatide, and tirofiban. These products may be given intravenously after percutaneous transluminal coronary angiography or an acute coronary syndrome, such as unstable angina or non-Q wave myocardial infarction (MI), to decrease the risk of vessel closure and further coronary

Secondary Prevention of Events [15]

Patient Subgroup	Recommended Agent	Secondary Agent[1]
post-MI	aspirin	clopidogrel
unstable angina	aspirin	ticlopidine
ischemic stroke	aspirin	clopidogrel
peripheral vascular disease	aspirin [2]	clopidogrel[2]

1 recommended if first choice is contraindicated or patient is allergic

2 Aspirin is a grade A1 recommendation (clear effect - benefits outweigh risks); clopidogrel is a grade A2 recommendation (effect equivocal - uncertainty whether benefits outweigh risks).

events.[9] Oral agents including xemilofiban, sibrafiban, orbofiban, lotrifiban, and lefradafiban are under investigation and may be marketed in the near future.

Two adenosine diphosphate inhibitors, ticlopidine and clopidogrel, are currently available for the secondary prevention of atherosclerotic events, including vascular death, stroke, and myocardial infarction.[10,11] Ticlopidine and clopidogrel irreversibly inhibit the binding of adenosine diphosphate to platelets, thereby preventing Glycoprotein IIb/IIIa receptor complexation and platelet aggregation.[9] Ticlopidine has demonstrated superior efficacy versus aspirin for stroke prevention in patients with a history of cerebral ischemia and peripheral artery disease.[12,13] Efficacy with ticlopidine has also been demonstrated in combination with aspirin following implantation of metallic coronary stents. This combination reduced hospitilization and bleeding complications versus traditional anticoagulation with warfarin.[14] A limiting side effect of ticlopidine is a risk of neutropenia observed in approximately 1.0% of patients treated.[15]

Clopidogrel was approved for secondary prophylaxis of myocardial infarction, stroke, and vascular death in patients with recent MI or stroke, or peripheral vascular disease in November 1997. At doses of 75 mg per day, clopidogrel irreversibly inactivates platelets, with inhibition beginning two to three days into therapy and maximal suppression occurring in four to seven days.[15] Following discontinuation of therapy, antiplatelet activity persists for seven to ten days corresponding with the plasma life of circulating platelets. Side effects that may occur with use of clopidogrel include rash, bleeding, diarrhea, GI upset, pruritis, and urticaria. The risk of neutropenia with clopidogrel is approximately 0.1%.[15,16]

Secondary prevention of cardiovascular events with clopidogrel was evaluated in a large trial ("A randomized, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE)") published in the *Lancet* in 1996.[11] Patients were assigned to treatment with either aspirin 325 mg/day or clopidogrel 75 mg/day. To be included in the trial, patients had to have one of three qualifying events including recent ischemic stroke (onset > 1 week and < 6 months before randomization), myocardial infarction (within 35 days of randomization), or atherosclerotic peripheral arterial disease.

> *Clopidogrel was approved for secondary prophylaxis of myocardial infarction, stroke, and vascular death in patients with recent MI or stroke, or peripheral vascular disease in November 1997.*

Average patient follow-up was 1.9 years. Patients in the clopidogrel group had an 8.7% relative risk reduction (95% CI 0.3-16.5, p=0.043) in a combined endpoint of ischemic stroke, MI, or vascular death when compared with the aspirin treated patients. There was not a statistically significant difference between treatment groups in the risk of death from any cause, vascular death alone, the cluster event of any stroke, MI, or death from any cause, or the cluster event of ischemic stroke, MI, amputation, or vascular death. For patients with peripheral arterial disease (n=6452), those receiving clopidogrel were less likely than those receiving aspirin to reach a first-outcome event of stroke, MI or vascular death (p=0.0028), with a relative risk reduction of 23.8% (95% CI 8.9-36.2). There was no significant difference between treatment groups for this outcome in patients with a previous stroke (n=6431) or MI (n=6302). Of note, the researchers found a statistically significant test of heterogeneity of these three treatment effects. This suggests that the benefit of treatment may not be similar among the three subgroups of patients (i.e., patients with a previous MI, stroke, or peripheral arterial disease).[11]

More patients treated with aspirin were likely to experience GI hemorrhage (2.66% vs. 1.99%, p<0.05) and indigestion/nausea/vomiting (17.59% vs. 15.01%, p<0.05) than clopidogrel-treated patients. The risk of any bleeding or intracranial hemorrhage was not statistically different between the two groups. Rash was more common among patients treated with clopidogrel (16.02% vs. 4.61%, p<0.05), as was diarrhea (4.46% vs. 3.36%, p<0.05).[11]

Based on the results of the CAPRIE trial, 196 patients would need to be treated with clopidogrel instead of aspirin in order to prevent one additional event of ischemic stroke, MI, or vascular death. (To put this into perspective, 125 patients need to receive simvastatin in order to prevent one death due to any cause in one year; 30 patients with chronic atrial fibrillation need to receive warfarin in order to prevent one embolic stroke in one year.) The cost, at average wholesale price, of treating 196 patients with clopidogrel for one year is approximately $215,000 (based on an AWP of $90 for 30 clopidogrel tablets). Aspirin yields a cost of $2300 to treat a similar number of patients. Therefore, it would cost an additional $212,700 to prevent one additional ischemic stroke, MI, or vascular event.

Aspirin is a cost-effective agent for the prevention of secondary atherosclerotic events and is the standard of care at this time. Clopidogrel has a role in the prevention of secondary events for patients with contraindications to aspirin given the reduced risk of neutropenia versus ticlopidine. Based on the results of the CAPRIE trial, patients with peripheral arterial disease will also benefit from the use of clopidogrel.[11] The American College of Chest Physicians' (ACCP) recommendations for the use of aspirin and clopidogrel are summarized in the table.[17] Currently, combination studies of clopidogrel with low-dose aspirin are ongoing. The emergence of new agents, such as oral Glycoprotein IIb/IIIa inhibitors, will likely impact the antiplatelet market in the future.

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Health by Numbers

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Jay S. Buechner, PhD, and William J. Waters, Jr., PhD

Use of Massachusetts Hospitals by Rhode Island Residents

Jay S. Buechner, PhD

For much of Rhode Island, there are hospitals in Massachusetts close enough to be alternatives to hospital care within the state. These hospitals include both the tertiary care referral hospitals in Boston and Worcester and community hospitals in Fall River, South Attleboro, and elsewhere. Information on the use of Massachusetts hospitals by Rhode Island residents is therefore relevant for monitoring health care utilization and disease patterns in the state's population. This report presents selected summary data on utilization of Massachusetts hospitals by Rhode Islanders during a recent five-year period.

Methods

Since October 1, 1989, acute care general hospitals in Rhode Island have reported information on every inpatient stay to the Department of Health's Office of Health Statistics per state regulation. The data file includes patient demographics, information on diagnoses and procedures, and data on charges and expected pay source. Similarly, hospitals in Massachusetts report discharge data to the Division of Healthcare Finance and Policy in the Massachusetts Office of Health and Human Services. Data on Rhode Island residents in the Massachusetts files for the period October 1, 1992, through September 30, 1997, corresponding to hospital fiscal years 1993 through 1997, were extracted using zip code of patient residence by HCIA, Inc., for the Department of Health. In the analysis of both data sources, discharges of all Rhode Island residents except hospital newborns were included. Percentages are made on combined discharges from Rhode Island and Massachusetts hospitals.

Results

During the five fiscal years 1993-1997, there were 32,203 non-newborn discharges of Rhode Island residents from Massachusetts hospitals, an average of 6,041 discharges per year. Annual utilization decreased from a high of 6,623 in fiscal year (FY) 1993 to 5,583 in FY1997. As utilization of in-state hospitals by residents decreased over this period also, the annual percentage of total discharges represented by Massachusetts hospitals varied only slightly, between 5.0% and 5.4%, from the five-year average of 5.3%.

During the same five-year period, the utilization of Rhode Island hospitals by Massachusetts residents declined more slowly, from 6,782 in FY1993 to 6,475 in FY1997. Thus, in FY1993, there were 102 discharges of Massachusetts residents from Rhode Island hospitals per 100 discharges of Rhode Island residents from Massachusetts hospitals, rising to a ratio of 116 in FY1997. Throughout the five-year period Rhode Island was thus a net exporter of hospital services to Massachusetts.

Rhode Islanders in the age group 45 - 64 years were most likely to seek care in Massachusetts facilities, and those ages 65 and older least likely. (Figure 1) Similarly, Rhode Island residents with certain principal diagnoses were more likely to be treated in Massachusetts, including those with congenital anomalies (25.4%), diseases of the nervous system (9.3%), neoplasms (7.8%), and diseases of the muscu-

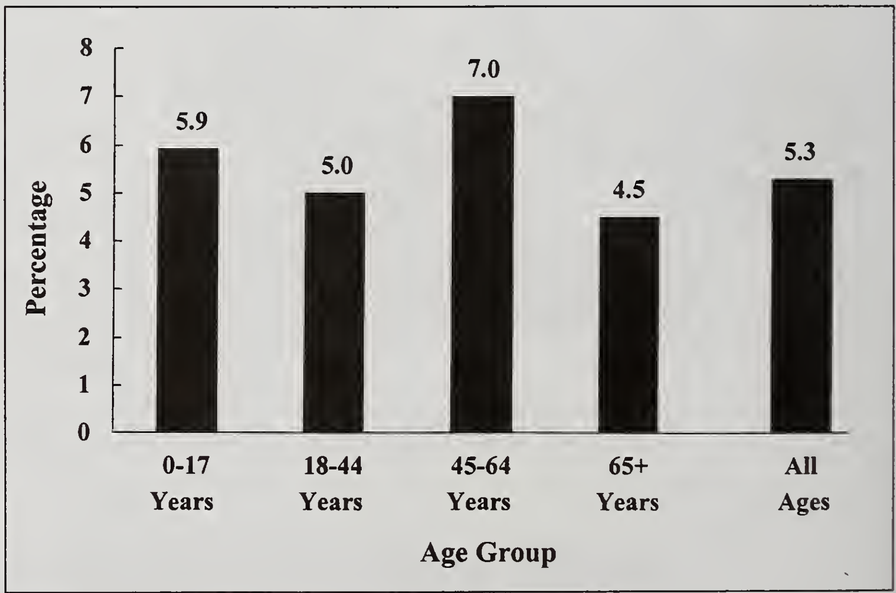

Figure 1. Percentage of Hospital Discharges of Rhode Island Residents Occurring in Massachusetts Hospitals, by Age Group, October 1, 1992 - September 30, 1997.

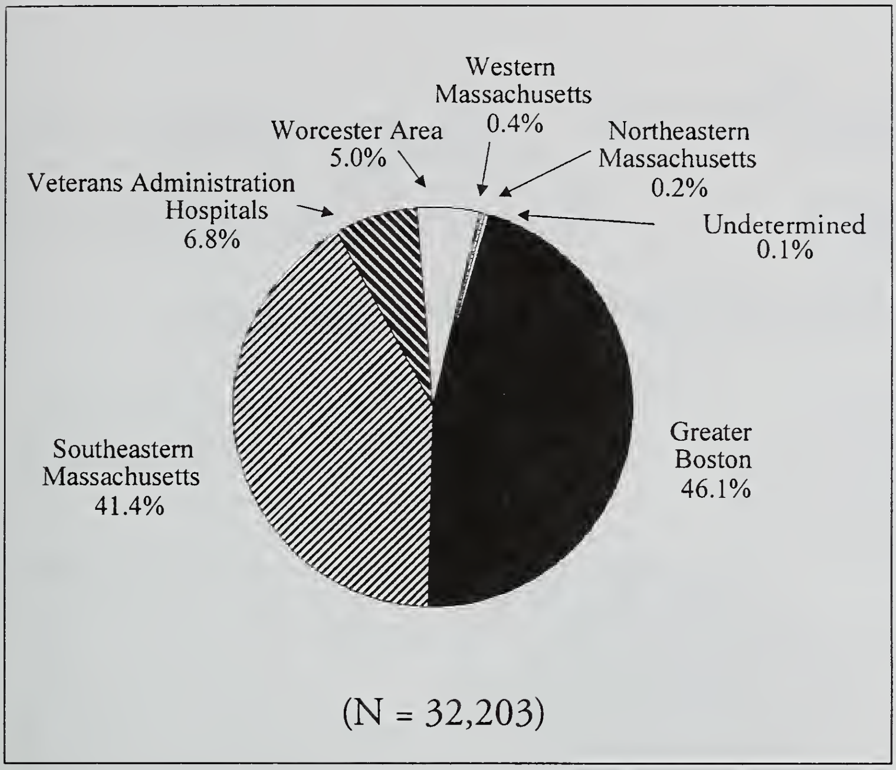

Figure 2. Hospital Discharges of Rhode Island Residents Occurring in Massachusetts Hospitals, by Location/Type of Hospital, October 1, 1992 - September 30, 1997.

loskeletal system (7.5%). Least likely to be treated in Massachusetts were those with diagnoses of pregnancy and complications (2.4%), diseases of the respiratory system (3.5%), infectious diseases (3.6%), and diseases of the skin and subcutaneous tissue (3.7%).

Residents of towns on or near the state's eastern border were most likely to seek care in Massachusetts hospitals. Tiverton (76.8%) and Little Compton (69.1%) residents are unique in their dependence on these facilities for care. Portsmouth (24.9%) and Warren (10.6%) are the only other towns whose residents receive at least 10% of their care in Massachusetts.

The largest number of discharges of Rhode Island residents were from hospitals in the greater Boston area (13,914), followed closely by southeastern Massachusetts facilities (12, 516). (Figure 2) Among the first group, Massachusetts General Hospital had the largest number of discharges (2,738), followed by Brigham and Women's Hospital (2,487), and Children's Hospital (1,747). In southeastern Massachusetts, Southcoast Health Systems' three hospitals, Charlton, St. Luke's, and Tobey, together had 6,378 discharges of Rhode Islanders, followed by St. Anne's Hospital, with 3,725 discharges.

Discussion

Annually, approximately 6,000 hospital inpatient stays of Rhode Island residents occur in Massachusetts hospitals, representing one of every twenty stays. In comparison, there were only 592 discharges of Rhode Island residents from Connecticut hospitals in FY97. The inclusion of data from Massachusetts hospitals is clearly important when calculating population-based hospital utilization rates for Rhode Island. Because of the observed variations, it is especially important to include these data when rates are calculated for specific diagnostic categories and for small geographic areas within the state.

Jay S. Buechner, PhD, is Chief, Office of Health Statistics, and Assistant Professor of Community Health, Brown University School of Medicine.

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PUBLIC HEALTH BRIEFING

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

Bicycle Helmets: It's the Law

Nancy Libby Fisher, MMHS

Head injury is the leading cause of death in bicycle crashes and is the most important determinant of bicycle-related death and permanent disability. Each year there are about 900 bicycle-related deaths in the United States, and hospital emergency rooms treat more than 500,000 people for bicycle-related injuries.[1]

The single most effective safety device available to reduce head injury and death from bicycle crashes is a helmet. The use of a helmet reduces the risk of bicycle-related head injury by up to 85% and the risk of brain injury by 88%.[1] Rhode Island State law requires helmet use for all bicyclists under age 16; the legislation does not impose a fine.

When the summer is over, many children will be riding their bicycles to school. To make bicycling fun and safe, the Rhode Island Department of Health is asking physicians to counsel patients and parents of young patients to wear helmets when riding bicycles. We encourage physicians to photocopy the attached page and make it available to patients and their families.

For more information on bicycle safety, please call the Unintentional Injury Program at the Rhode Island Department of Health, (401) 222-4420.

REFERENCES

1. National Safe Kids Campaign. *Fact Sheet.* December 1998. (1301 Pennsylvania Avenue, Washington, DC)

Nancy Libby Fisher, MMHS, is Program Manager of the Rhode Island Unintentional Injury Prevention Program at the Rhode Island Department of Health.

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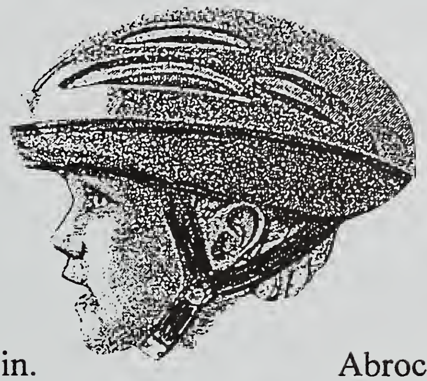
The helmet should fit snugly.
Use the foam pads to make it fit.

The helmet should cover the forehead.

The helmet should be level on the head.

The ear glides should be put just below the ears.

Fasten the strap under the chin.
Always buckle the strap.



- **Safety Tips**

Always ride on the right with the flow of traffic.

Obey traffic signs and signals.

Use hand signals.

Be sure the bicycle is the right size.

Do not ride two people on a bicycle.

Stop and look for traffic before riding into the street from a driveway.

- **Questions: 401-222-4420**

Salva Vidas.
Use el Casco.

Es la ley.

El casco debe quedar bién ajustado.
Use las esponjas para ajustarlo.

El casco debe cubrir la frente.

El casco debe quedar al nivel de la cabeza.

Las hebillas del casco deben ponerse debajo de las orejas.

Abroche la correa debajo de la barbilla.
Abroche la correa todo el tiempo.

- **Consejos de Seguridad**

Mantenerse a la derecha del tráfico todo el tiempo.

Obedecer las señales de tráfico.

Usar señales con la mano.

Asegúrese que la bicicleta es el tamaño correcto.

No anden dos personas en la misma bicicleta.

Antes de salir a la calle para un paseo en bicicleta, hay que parar y mirar si vienen carros.

- **Preguntas: 401-222-4420**

Judicial Diagnosis

The Good Samaritan Physician

Lawrence W. Vernaglia, JD, MPH

Under what circumstances will a physician or other health care professional in Rhode Island be liable for injuries caused by the professional's efforts to render first aid or other emergency assistance to a person in need? This essay will answer that question and address the legal and ethical implications of Rhode Island's "Good Samaritan" laws.

The common law principle applicable in American courts is that "one who assumes to act, even though gratuitously, may become subject to the duty of acting carefully if he acts at all."[1] Absent a specific statute to the contrary[2] or some prior special relationship with the person — such as a preexisting physician-patient relationship — a physician has no legal duty to render emergency assistance to a person in need even if the assistance could be rendered with minimal or no cost or risk to the physician.

However, physicians are ethically bound to render emergency care to another under reasonable circumstances. The Code of Medical Ethics of the American Medical Association teaches that although physicians are free to choose whom they will serve, "[t]he physician should, however, respond to the best of his or her ability in cases of emergency where first aid treatment is essential. Once having undertaken a case, the physician should not neglect the patient." [3]

> *The effect of the Good Samaritan Law is that physicians, nurses, or any other persons who in good faith and without pay undertake to perform cardiopulmonary resuscitation or the Heimlich maneuver, reverse an allergic reaction, stop profuse bleeding, or administer any other emergency first aid will not be liable to the victim even if the rescuer performs his duties "negligently."*

Thus, the common law, which was followed in Rhode Island, established a framework in which physicians were not legally required to come to the assistance of an accident victim; but if they did, they must exercise due care or be liable for any deviations which caused the victim injury. There have been few, if any, reported cases assigning liability to the doctor in such circumstances. This is likely because courts are hesitant to establish a disincentive for talented medical professionals to render emergency care or first aid when needed. Despite that fact, many professionals fear liability - a fear that has deterred some from rendering care in a situation where they might otherwise have done so.

As a result, many states, including Rhode Island, passed so-called "Good Samaritan" laws that shield a practitioner from liability for ordinary negligence when rendering gratuitous emergency care. Rhode Island's statutes do just that, and are broader than most other states' laws. Rhode Island General Law § 9-1-27.1 reads: "No person who voluntarily and gratuitously renders emergency assistance to a person in need thereof, including the administration of life-saving treatment for those persons suffering from anaphylactic shock shall be liable for civil damages which result from acts or omissions by such persons rendering the emergency care, which may constitute ordinary negligence. The immunity does not apply to acts or omissions constituting gross negligence or willful or wanton conduct."[4]

The effect of the Good Samaritan Law is that physicians, nurses, or any other persons who in good faith and without pay undertake to perform cardiopulmonary resuscitation or the Heimlich maneuver, reverse an allergic reaction, stop profuse bleeding, or administer any other emergency first aid will not be liable to the victim even if the rescuer performs his duties "negligently." Negligence is a legal term that means a deviation from the standard of care that a reasonably prudent and careful person would use under similar circumstances. Medical malpractice cases are customarily about negligence in the performance of medical services by a physician. The Good Samaritan Law does not protect the rescuer from liability if the emergency assistance is rendered so incompetently as to constitute "gross negligence or willful or wanton" conduct. Gross negligence or willful or wanton conduct connotes a much higher degree of error and culpability. These terms implicate an act or omission breaching a legal duty of an aggravated character as distinguished from a mere failure to exercise ordinary care. A simple mistake in providing care will not rise to the level of gross negligence or willful or wanton conduct.

Rhode Island courts have had few opportunities to interpret, and even fewer to apply, the Good Samaritan laws. In each case, the physician or other person rendering voluntary aid was not held liable for the injuries. A physician and a nurse voluntarily staffing the first aid station at the 1989 Cajun Music Festival in Escoheag, Rhode Island recently invoked the Good Samaritan laws. Ralph Boccasile was attending the festival when he went into anaphylactic shock[5] subsequent to consuming a quantity of seafood gumbo. Dr. Sarah John, a past president of the American Association of Women Emergency Physicians, came to Mr. Boccasile's assistance and attempted to treat the allergic reaction with an Epipen. Mr. Bocassile was eventually brought to a hospital. Unfortunately, the rescue efforts were unsuccessful. Mr. Boccasile's widow brought suit against Dr. John, the nurse, and other defendants for negligence in not responding adequately to her husband's allergic reaction. Two separate judges of the Rhode Island Superior Court concluded that the protections of the Good Samaritan laws immunized Dr. John and her nurse from liability. On appeal, the Rhode Island Supreme Court, too, found Dr. John and her nurse not liable in the death, although the Supreme Court did not base its decision on the Good Samaritan Law *per se*, but, rather, on the fact that the widow had introduced no expert medical evidence to establish that the volunteers' actions had deviated from the standard of care.

One interesting lesson from the Boccasile case is the extent to which courts are willing to determine that a doctor is acting as a "volunteer." At the Cajun Music Festival, Dr. John was staffing the first aid booth in an arguably professional context, although she was not receiving any financial remuneration that we know of. The issue is open as to whether receipt of free admission or discounts at a program such as the festival might constitute sufficient remuneration to render the physician services no longer "voluntary." It is apparent that the Superior Court judges were unwilling to ascribe a greater duty to the physician even though she was acting as an "official" volunteer at the location and in an identifiable role as a care-giver to the patrons of the festival. Other states have grappled with how far to extend this logic. The Utah Supreme Court, interpreting the Utah Good Samaritan law which is nearly identical to the Rhode Island statute, held that a physician employed by a hospital is entitled to immunity in responding to in-house emergencies, unless he or she has a pre-existing duty to do so.[6] Bills have been introduced in California to provide doctors with immunity from negligence when volunteering at homeless shelters, and to waive the requirement of maintaining malpractice insurance in such contexts. Naturally, patient advocates object to establishing laws that provide the poorest patients with no redress for negligent care.

In another recent case, a British physician, Dr. John Stevens, responded to a call for a doctor on an intercontinental flight. Dr. Stevens correctly diagnosed a dangerous blood clot in a fellow passenger and reported that he ministered to her until the flight was grounded in Chicago and the patient transferred to an appropriate medical facility. The airline gave Dr. Stevens a bottle of champagne and a $250 travel voucher in thanks for his services. Dr. Stevens later complained that his services were worth more than a "bottle of cheap champagne" and he sent a bill to the airline for £540 or about $900. [7] He then sued the airline in small claims court to collect on the debt. Whatever this story may have to say about the status of famed British courtesy, if the case had been subject to the Rhode Island Good Samaritan Law, we would note that at the time Dr. Stevens rendered his services on the airplane, the services were apparently voluntary, and he would have been immune from ordinary negligence were damages to result from his actions. Indeed, even after he received and accepted the champagne and travel voucher, he still would likely have been deemed a volunteer because he was not expecting the gifts at the time he performed the services.

However, upon his submission of a bill for $900, his work was transformed from gratuitous services to activities for which the doctor expected a fee. If the patient were to discover an injury caused by the negligence of Dr. Stevens, he would no longer receive immunity from negligence. This case also illustrates the difficulty in determining which law applies in such circumstances. This is significant because Good Samaritan laws vary from state to state. Some statutes only protect physicians; others, such as in Rhode Island, protect

everyone. Some state laws provide for immunity only at the scene of a motor vehicle accident, others, again such as in Rhode Island, protect all good faith gratuitous rescues. Ordinarily, the law in effect where the services are rendered will apply. In a case involving alleged negligence onboard an airplane, a court will have to determine which jurisdictions' laws to apply. The choices include the state where the flight originated or the destination, or, conceivably, the state over which the acts transpired.

In summary, physicians, nurses, and other persons who come to the aid of a person in an emergency, without expectation of any compensation, and without a prior duty to care for such a person, are, in Rhode Island, immune from damages even if the care is provided negligently and damages to the victim are proximately caused thereby. In other states, similar laws apply - but in most reported cases, regardless of the statute, a volunteer physician is usually spared liability from negligence.

END NOTES.

1 Therrien v. First Nat. Stores, 6 A.2d 731, 63 R.I. 44 (1939); see also, Restatement (Second) of Torts § 323 (1965).

2 Some states affirmatively require a physician or other person to come to the rescue of a person in need. See e.g., Minnesota Statutes § 604A.01 (1996) and Vermont Statutes § 12-23-519 (1997). Rhode Island does not impose such a legal duty on its citizens, absent some pre-existing duty.

3 American Medical Association Code of Medical Ethics, Current Opinions with Annotations § 8.11.

4 As indicated, this statute is more expansive than many other state's laws because it applies to all persons not just physicians. Rhode Island has other more specific Good Samaritan laws granting immunity from liability for volunteers who perform CPR or employ automated external defibrillators (AEDs), provided that the volunteer has been trained under American Heart Association for Red Cross standards. R.I.G.L. § 9-1-34. As of June 18, 1999, a recent amendment to the law extends the immunity to certain educators teaching CPR or AED use. There is also a Good Samaritan law applicable to the police and firefighters. R.I.G.L. § 9-1-27. Interpreting the interplay between these laws is beyond the scope of this essay, although for purposes of trained medical professionals, reliance on the primary Good Samaritan Law is sufficient.

5 The language regarding anaphylactic shock in the Good Samaritan Law was added after the events at the festival transpired.

6 Hirpa v. IHC Hospitals, Inc., 948 P.2d 785 (Utah, 1997).

7 J. Lynn Lunsford, Doctor Who Helped Save Passenger's Life Bills Airline for Service, *Dallas Morning News*, October 3, 1998.

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Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	August 1998	12 Months Ending with August 1998		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	247	3,140	317.1	3,793.5
Malignant Neoplasms	192	2,522	254.7	6,873.5*
Cerebrovascular Diseases	43	647	65.3	742.0
Injuries (Accident/Suicide/Homicide)	32	347	35.0	7,003.0
COPD	32	475	48.0	395.0

Vital Events	Reporting Period		
	February 1999	12 Months Ending with February 1999	
	Number	Number	Rates
Live Births	920	13,445	13.6*
Deaths	887	9,687	9.8*
Infant Deaths	(5)	(99)	7.4#
Neonatal deaths	(4)	(74)	5.5#
Marriages	350	7,444	7.5*
Divorces	323	3,203	3.2*
Induced Terminations	380	4,613	343.1#
Spontaneous Fetal Deaths	47	1,055	78.5#
Under 20 weeks gestation	(41)	(962)	71.6#
20+ weeks gestation	(6)	(93)	6.9#

* Rates per 1,000 estimated population # Rates per 1,000 live births

*Excludes one death of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 990,225

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

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Content:

- ♥ Lipid Management
- ♥ Stress Management
- ♥ Hypertension
- ♥ Behavior Modification
- ♥ Special Issues in Minority Population
- ♥ Exercise
- ♥ Weight Loss
- ♥ Smoking Cessation
- ♥ Secondary Prevention of Heart Disease

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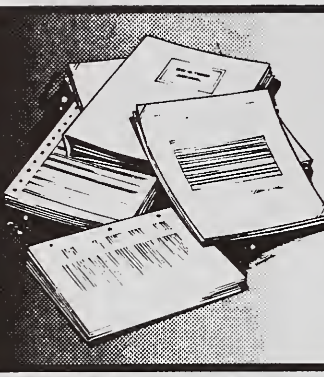

CLINICAL TRIALS DIRECTORY

Medicine & Health/ Rhode Island is pleased to launch this Directory. If you would like to list a trial, please contact: Joan Retsinas, Managing Editor.
phone: (401) 272-0422
fax: (401) 272-4946
e-mail: JRetsinas@aol.com.

Pharmacotherapy of body dysmorphic disorder

Sponsor: National Institutes of Mental Health

Purupose: Evaluate the effectiveness of psychotropic medication for body dysmorphic disorder, a distressing and impairing preoccupation with a nonexistent or slight defect in appearance. This study is geared for adolescents and adults.

Principal Investigator: Katharine A. Phillips, MD

Patients Recruited: Age 12 to 65, both genders, no unstable medical problems, no history of seizures

Intervention: Patients receive a comprehensive psychiatric and medical evaluation. They are treated with medication which appears effective for body dysmorphic disorder. All evaluation and treatment are free of charge. For patients who participate in a placebo-controlled trial, patients randomized to placebo subsequently receive medication at no charge.

Duration of study: Three months

Phase: 4

Site: Butler Hospital

Remuneration for patients: None; however, all evaluations and treatment are provided free of charge.

Contact: Kelly Viens; phone: (401) 455-6466; fax: (401) 455-6539; e-mail: Katharine_Phillips@brown.edu

A Multi-Center, Placebo-Controlled Trial of Melatonin for Sleep Distrubance in Alzheimer's Disease

Sponsor: Alzheimer's Disease Cooperative Study

Purpose: Brian Ott, MD, is conducting this trial to treat Alzheimer's Disease patients who experience sleep disturbances associated with Alzheimer's Disease. Two doses of melatonin will be used to treat sleep disturbances in order to lessen the burden on caregivers and family members.

Patients Recruited: Anyone 55 years or older with a diagnosis of probable Alzheimer's Disease experiencing sleep disturbances.

Intervention: Two doses of Melatonin vs. placebo

Duration of study: 12 weeks

Phase: III

Site: Alzheimer's Disease & Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860

Contact: Michael Pimental, phone (401) 729-3752

Earlier vs. Later Levodopa in Parkinson's Disease (ELLDOPA)

Sponsor: NIH and the Parkinson Study Group

Purpose: Joseph Friedman, MD, is conducting this trial to compare the effect of early or late treatment of Parkinson's Disease with L-Dopa to answer the question of whether L-Dopa slows or hastens the progression of Parkinson's Disease.

Patients Recruited: Patients must be 30 years or older, must be diagnosed with Parkinson's Disease within the last 2 years. No eldepryl, L-DOPA, amantadine, anticholinergics, antihistamines, antidepressants, or benzodiazepines for previous 30 days.

Intervention: varying doses of Carbidopa/Levodopa vs. placebo

Duration of study: 9 months

Site: Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860

Contact: Margaret Lannon, RN, MS, phone: (401) 729-3750

A Phase III Multicenter, Double-Blind, Parallel-Group Placebo Controlled Study of the Effect of Riluzole 50 mg BID or 100 mg BID for Two Years on the Progression of Parkinson's Disease in 1050 Patients

Sponsor: Rhône-Poulenc Rorer

Purpose: The purpose is to treat recently diagnosed patients with the drug Riluzole, in order to study the effect this drug has on delaying the progression of Parkinson's Disease.

Principal Investigator: Joseph H. Friedman, MD

Patients Recruited: Patients must have recently diagnosed Parkinson's Disease with symptoms present for no more than 3 years and must currently be receiving no medications to treat Parkinson's Disease.

Intervention: Two doses of Riluzole or Placebo

Duration of Study: 2 years

Phase: III

Site(s): Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860

Contact: Margaret C. Lannon, RN, MS; phone: (401) 729-3750

Psychosis and Associated Behavioral Disturbances in Patients with Dementia

Sponsor: a major pharmaceutical company

Purpose: The purpose is to determine whether persons with psychosis and behavioral disturbance related to

Alzheimer's Disease or vascular dementia benefit from antipsychotic drugs.
Patients Recruited: Patients must be at least 40 years old and have hallucinations or delusions associated with dementia caused by Alzheimer's Disease or stroke.
Intervention: One of two antipsychotic drugs, or Placebo
Duration of Study: 28 weeks
Phase: III
Site(s): Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860
Contact: Michael Pimental, phone: (401) 729-3752

A Randomized, Double-Blind Placebo-Controlled Trial to Evaluate the Safety and Efficacy of Vitamin E and Donepezil HC1 (Aricept®) to Delay Clinical Progression to Alzheimer's Disease (AD) from Mild Cognitive Impairment (MCI)
Sponsor: Alzheimer's Disease Cooperative Study and Pfizer/Elsai
Purpose: The purpose is to determine whether treatment of persons with mild forgetfulness can prevent the actual development of Alzheimer's Disease or slow the progression of Alzheimer's Disease
Principal Investigator: Brian R. Ott, MD
Patients Recruited: Patient must be 55 to 90 years of age and have memory difficulties that are not severe enough to interfere with carrying out one's usual daily activities (MMSE=24-30). Must be willing to take only vitamin supplement provided.
Intervention: Multivitamin, Vitamin E, or Donepezil HC1
Duration of Study: 3 years
Phase: III
Site(s): Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860
Contact: Michael Pimental, phone: (401) 729-3752

The Effect of Pramipexole on Depressive Symptoms in Parkinson's Disease Patients
Sponsor: Pharmacia and Upjohn, Inc.
Purpose: To evaluate the effectiveness of pramipexole on the depression and other non-motor symptoms in patients with Parkinson's Disease
Principal Investigators: Joseph H. Friedman, MD, and Hubert Fernandez, MD
Patients recruited: Men and women with PD, 30 - 85 years old, who have taken no dopamine agonist medications in the past 3 months and are clinically depressed
Intervention: Pramixpexole or placebo
Duration of study: 19 weeks
Phase: IV
Site: Movement Disorders Unit, Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860
Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

Pilot Huntington At Risk Observational Study (PHAROS)
Sponsor: Huntington Study Group (HSG)
Purpose: To observe individuals who are considred "at risk" for developing Huntington's disease (HD) because they have (or had) a parent or sibling with HD. Participants will be examined every 9 months for at least 3 years.
Principal Investigators: Joseph H. Friedman, MD, and Hubert Fernandez, MD
Patients recruited: "At risk" individuals 30 - 54 years old who have never been (and have no desire to be) tested for the HD gene, and who have no definite signs or symptoms of the illness.
Intervention: None
Duration of study: At least 3 years
Site(s): 38 centers in the United States and Canada. Locally at Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860
Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

A Randomized , Double-Blind, Placebo-Controlled Study to Determine the Safety and Efficacy of FK960 in Patients with Mild to Moderate Probable Alzheimer's Disease
Sponsor: Fujisawa Research Institute of America, Inc.
Purpose: The purpose is to assess the safety and efficacy of four different doses of FK960 vs. placebo in patients with mild to moderate probable Alzheimer's Disease and to identify at least one dosage level of FK960 effective in improving symptoms of Alzheimer's Disease
Principal Investigator: Brian R. Ott, MD
Patients recruited: Patients 55 years or older with probable Alzheimer's Disease who are presently not receiving drug treatment for Alzheimer's Disease and who are not presently taking antidepressants or tranquilizers
Intervention: FK960 or placebo
Duration: 6 1/2 months
Phase: II
Site: Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860
Contact: Michael Pimental, phone: (401) 729-3752

***The Effect of Riluzole on the Progression of Parkinson's Disease in Patients Treated with L-Dopa or Dopamine Agonist**

Sponsor: Rhône-Poulenc Rorer

Purpose: To demonstrate that riluzole slows the progression of Parkinson's Disease by showing that treatment with riluzole delays changes in symptoms that necessitate changes in treatment.

Investigators: Joseph H. Friedman, MD, and Hubert H. Fernandez, MD

Patients Recruited: Individuals who have had Parkinson's Disease for less than 5 years and have been treateed for less than 3 years with L-Dopa or a dopamine agonist.

Intervention:Riluzole 100 mg or 200 mg per day or placebo

Duration: 1-2 years

Phase: III

Site: 130 centers throughout the world. Locally at: Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860

Contact: Margaret C. Lannon, MR, MS, phone: (401) 729-3750

*** A Double-Blind, Placebo-Controlled Study of a Vaccine to Prevent Cancer caused by HPV***

Sponsor: a pharmaceutical company

Purpose: To determine the efficacy of a vaccine to prevent cancer caused by the Human Papilloma Virus (HPV)

Investigator: Dr. Pauline Wood

Patients Recruited: Women 16-23 years of age who are sexually active or plan to be sexually active

Intervention: Some patients will get the vaccine; others will get the placebo.

Duration: 2.5 years

Phase: II

Site: Potter Building, Health Services, University of Rhode Island

Remuneration for Patients: Qualified psrticipants will receive free gynecolocial exams, free STD testing, and financial compensation up to $650 for time and travel

Contact: Janet Kropp, Coordinator, phone: (401) 782-2570; fax: (401) 874-2586

*** Zyban Collaborative Study***

Sponsor: National Institutes of Health; The Miriam Hospital, a Lifespan partner; and Butler Hospital, a Care New England partner

Purpose The Zyban Collaborative Study is investigating the effects of the combination of Zyban, an FDA-approved medication for smoking cessation, and group counseling to assist with quitting smoking. It is anticipated that this combination of treatments will be particularly effective in the increasing number of "hard core" smokers.

Principal Investigator: Raymond Niaura, PhD, and Richard Brown, PhD

Patients recruited: This free research study is open to cigarette smokers who are at least 18 years old, in good health, smoke 10 or more cigarettes a day, and want to quit smoking.

Intervention: Participants have a 50/50 chance of being assigned the active medication Zyban versus a placebo; they will receive a free medical screening and 12 group counseling sessions.

Duration of study: Participation in the study will last up to one year and will include 12 group visits during a 3-month treatment period, and 3 follow-up visits at 2, 6, and 12 months after the start of treatment.

Phase: Subject recruitment will continue for approximately two-and-a-half years. Participant screenings are scheduled on a continuous basis; new groups begin every seven weeks. Evening groups are available.

Site(s): The Miriam Hospital and Butler Hospital

Remuneration for patients: Participants will receive up to $140 for completing all of the questionnaires and follow-up assessments after treatment ends. Abstinence from smoking is not required in order to be reimbursed.

Contact: The Lifespan Health Connection for further information and screening for participation in the study. phone (401) 444-4800 Dr. Elizabeth Lloyd with specific questions. phone: (401) 793-3714, e-mail: ELloyd@Lifespan.org

Developmental Manifestation of Daytime Sleepiness

Sponsor: National Institute of Nursing Research

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THE RHODE ISLAND MEDICAL JOURNAL

The Official Organ of the Rhode Island Medical Society
Issued Monthly under the direction of the Publications Committee

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NINETY YEARS AGO

[AUGUST, 1909]

An editorial praised the completion of Providence City Hospital and its newly-chosen superintendent, Dr. D.L. Richardson (formerly assistant superintendent of Rhode Island Hospital). The City Council would need to act for this hospital to care for advanced cases of tuberculosis.

A second editorial lauded the compulsory notification of cases of tuberculosis, recently required by law. As for the voices of complaint protesting the "red tape" of the regulation, the editorial promised that the voices of complaint "will not be the voices of relieved victims nor of citizens who ...are protected."

Charles H. Baily, MD, reported on "Case of a Sudden Death following Lavage of the Stomach." A 40 year-old housewife had complained of nausea, vomiting, and headache for three weeks. Using a tube, the physican washed her stomach with a sodium bicarbonate solution until the fluids returned clear. Immediately afterward, the patient felt weak. Forty minutes later, she collapsed, unconscious and cyanotic.

Charles Chapin, MD, reported on the Health of Providence for June and July, 1909: 49 cases of scarlet fever (no deaths), 53 of diphtheria (6 deaths), 13 of typhoid fever (5 deaths), 103 of measles (17 deaths).

FIFTY YEARS AGO

[AUGUST, 1949]

Tom D. Spies, MD, the Director of the Nutrition Clinic, Hillman Hospital, Birmingham, Alabama, and Professor of Nutrition and Metabolism, Northwestern University Medical School, delivered the Eighth Annual Charles V. Chapin Oration: "The Control of Pellagra." Dr. Spies traced pellagra to 1735, when Gaspar Caspal, physician to King Philip V of Spain, recorded "mal de la rosa" among peasants. Lombardy peasants later coined the term to denote "rough skin." By manipulating the diet of "pellagrens" at the Institition for Insane Negroes in Alabama [with dried brewers yeast, liver extract, vitamins, nicotinic acid], researchers concluded, "Vitamin therapy alone is not sufficient...viamins cannot take the place of a well-balanced diet (3000-4000 caolories, 120-150 grams of protein)."

Louis E. Burns, MD, discussed "Hypoglycemia: Prevention, in the new Diabetic, with early Stabilization on Low Fat Diet." He reminded readers that "...the first physician that treats the new diabetic is the master of his or her destiny." He urged the value of small doses of insulin: "the taking of these small doses made the patient realize that he was still a diabetic, and had a psychological value in making him follow the treatment faithfully....Most diabetics, when cut loose from restrictions, go adrift..."

The Rhode Island Medical Society announced its new 24-hour medical bureau, a secretarial exchange for physicians that would refer emergency callers to physicians.

TWENTY FIVE YEARS AGO

[AUGUST, 1974]

In a Message from the Dean, Stanley Aronson, MD, discussed "The Future of Medical Education in the United States," as predicted by 111 deans of North American medical schools. Specifically, they predicted more women students (from12.8% in 1972-73 to 30% in the 1980s), the disappearance of "voluntary teaching," and a greater role for states in paying for medical education. A strong majority (87%) saw a National Health Insurance system on the horizon.

Thomas P. Hackett, MD, and Ned H. Cassem, MD, discussed "The Impact of Myocardial Infarction," urging readers to pay attention to psychosocial factors that induce depression during convalescence. (This paper was excerpted from a talk read at the 163rd Annnual Scientific Assembly of the Rhode Island Medical Society).

Michael G. Pierik, MD, in "Acute bone marrow suppression following 1-day oral administration of methotrexate," described the case of a 52 year-old male who took 15 mg of methotrexate for treatment of rheumatoid arthritis. Four days after discharge, the patient had increasing nausea, oral soreness, weakness, and diarrhea. Dr. Pierik urged methotrexate as a recourse "only following failure of conventional therapy."

Rev. Joseph Lennon, OP, (Vice President for Community Affairs, Providence College) in "The God Squad: Life should be safeguarded and promoted at all levels no matter what its condition," delineated ordinary from extraordinary measures and "the principle of the double effect."

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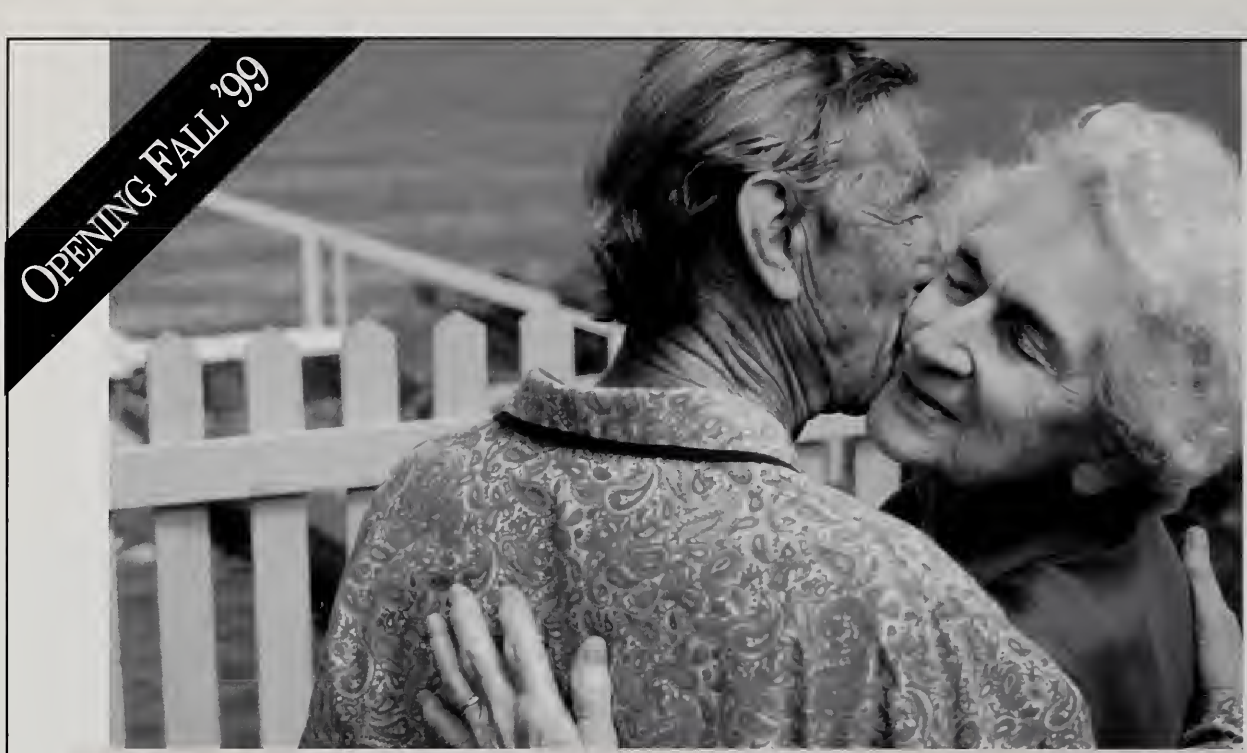

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Cover: "*Nimble Sewers,*" oil painting, by Paul R. Olson, a Riverside artist whose scenes of mall construction hang in Providence Place.

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COMMENTARIES

Arthritis and the Mission of *Medicine & Health/Rhode Island*

This Journal has been in existence over 100 years, but only recently survived a near death experience. Since I became editor-in-chief in January, we have put out nine issues, one per month, as the journal always has. However, the Journal has had to undergo some major changes for this to happen and hopefully to remain vigorous. It is hoped, of course, that each change will be for the better. I believe that most readers do not know how the Journal functions and may also wonder what is the Journal's function.

Medicine & Health/ Rhode Island (MH/RI)has undergone several changes over the years. It is no longer solely a publication of the Rhode Island Medical Society. MH/RI is subsidized by the Rhode Island Medical Society (RIMS), Brown University School of Medicine, Rhode Island Quality Partners, the American College of Physicians, and the Rhode Island Department of Health. In recent years each entity, except for the RIMS, provided an annual fixed subvention for the Journal. Whatever shortfall occurred by the year's end was picked up by the RIMS. RIMS, having no control over the Journal's budget, and worried about potential (and occasionally real) overspending, voted to divest itself of the Journal, but was persuaded to give it another year's grace period. The RIMS position was that it would like the Journal to continue, but only if a ceiling could be placed on the Society's financial risk. With this in mind I began my tenure.

We convened a new group to function as a "publication committee" of the Journal to provide oversight, both economic and critical. The committee was made up of representatives of the supporting bodies, each represented according to its allocation. This group meets quarterly to review the finances and the direction of the Journal, certifying that it is meeting its mission, and suggesting new ideas for the Journal to pursue. The committee adopted the following mission statement:

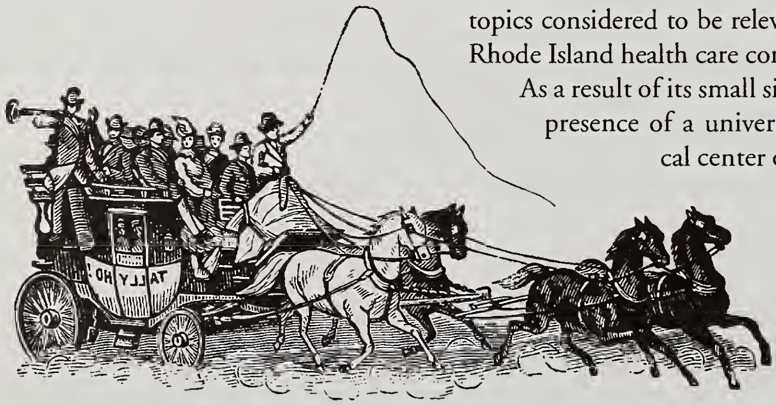
Medicine & Health/Rhode Island is intended to serve the medical and health community of the state of Rhode Island by providing a venue for scholarly, peer-reviewed articles that focus on issues of direct relevance to Rhode Island.

These articles will highlight medical and public health problems, local expertise, organizations, programs, epidemiology, quality assessments, outcome measures, education and any other topics considered to be relevant to the Rhode Island health care community.

As a result of its small size and the presence of a university medical center of international stature, this state is uniquely positioned to provide articles that bridge the gap between the research and practice-oriented parts of the community. It is the aim of this Journal to meet the above goals while bridging this gap.

This issue of the Journal is devoted to arthritis, a disorder of major importance to a large percentage of our population. The Department of Health, filling its mandate from a recently obtained grant, is helping to educate RI physicians by underwriting the mailing of this issue to every physician in the state. This is the second issue this year, the asthma (July) issue being the other, that has been mailed to every RI physician. The asthma issue was financed by the Robert Woods Johnson Foundation. We view this as a sign of success in our mission. We are using local expertise to educate our local health care community on local problems. And we are doing so within narrow financial constraints. Future issues will deal with driving, nutrition, adolescent medicine, family medicine, spinal cord injury, sports medicine and a variety of other topics. We welcome your comments, your scholarly contributions, and your continued support.

– Joseph H. Friedman, MD



The expanded mailing of this special edition is supported by the Rhode Island Department of Health, Arthritis Prevention and Management Program. For more information, please call (401) 222- 3496.

A Frenzied Passion in the Head

"As far back as I can remember I was a martyr to headache. No doctor could help me, no one seemed to know the cause. It was migraine, and though I watched it carefully I could not trace it to any fault of mine." So wrote Max Muller in 1899. He was Britain's most illustrious scholar concerning Sanskrit and Oriental languages. But none of his memorable textbooks or brilliant analyses could provide him with any semblance of relief when the periodic attacks of migraine overtook him.

There are many forms and varieties of headache but the term migraine should be reserved for those repetitive, unrelenting headaches of many hours duration with the following additional characteristics: They are typically severe, often unilateral, frequently preceded by visual auras, accompanied regularly by nausea and vomiting, and often require bedrest; and they are generally succeeded by an interval of depressed moodiness and pervasive fatigue. The disease was recognized and meticulously described by Hippocrates, Galen and Aretaeus. Because of its tendency to involve only one half of the head they called it hemi-cranium and over the centuries the word was corrupted to micranium and today, migraine. Somewhere along the line arose the word, megrim, which persists today as a synomyn not only for migraine but also for feelings of general sadness and lassitude ['the megrims.'] The Victorian era vapours and the sick headaches of the 1920s probably were migrainous episodes.

About 2 to 7% of the American population are said to be active victims of migraine. A careful survey in England suggested a somewhat higher prevalence of 10%. All surveys, though, have concluded that women are far more vulnerable than men to this form of headache. The onset typically is at puberty. The recurring headaches then increase in intensity reaching their height in the third decade of life and tend then to diminish as the senior decades are neared.

Like gout, migraine has always been cherished as a disease of distinction, an illness reserved for those with heightened intelligence. In the 17th Century migraine was said to affect those engaged in excessive thinking but with little physical exercise. So many scholars, clerics and upper class women suffered from migraine that *la vie spirituelle* was deemed a prerequisite before diagnosing the disease. By the 19th Century a migraine personality emerged. Such individuals were alleged to be obsessively rigid, annoyingly neat and punctilious. Recent epidemiologic studies in Great Britain seem to give credence to the notion that the illness is substantially more common in the higher socioeconomic strata.

Many migraine victims can predict the onset of their attacks by characteristic premonitory warnings which regularly precede the impending headache. Sometimes this alerting aura consists of a feeling of inappropriate lassitude but generally it involves the eyes with focal blurring of vision or temporary blind spots each surrounded by shimmering, brilliant lights of varying color and intensity; sometimes, too, there may be a transient blindness in one eye. These displays can be initially frightening but migraine victims become resigned to such spectral displays as they come to appreciate their transitory nature. The remarkable 12th Century Benedictine abbess, Hildegard of Bingen, endured many such personal experiences, describing episodes consisting of "multitudes of sparks" accompanied by "lights more brilliant than the sun." Given the rudimentary development of neurophysiology in the 12th Century, it is not surprising that an agile, spiritually oriented mind such as Hildegard's might interpret these visual auras as derived from divine authority rather than from a banal source such as a short-lived intracranial derangement.

Still other migraine victims, immediately prior to an attack, experience a temporary change in how they perceive their own bodies. Charles Dodgson, known better by his pseudonym, Lewis Carroll, was a lifelong victim of migrainous headaches; and he inserted some of his distressing perceptions of body shrinkage into the story line of Alice entering a wonderland. A still rarer prodrome preceding an attack of migraine is the disconcerting delusion of seeing a ghostly image nearby while knowing full well that it is not real. Linneaus, the Swedish physician who established the binomial system of naming all plants and animals, was a victim of severe migraine headaches. He had experienced, at times, the curious visual delusion of seeing himself nearby which warned him of an impending attack.

There has never been a shortage of theories regarding the cause of migraine. Some considered it the result of some gastrointestinal mischief, a gallbladder disorder perhaps, with disbalance of the vital juices. Others were convinced that those with hypersensitive brains became uniquely vulnerable to the baneful influence of febrile thoughts, a form of cerebral allergy. Those with less lofty thoughts blamed migraine on ill-defined toxins generated in the lower colon. And still others perceived the attacks as evidence of some gonadal derangement. A common explanation blamed migraine upon excessive reading with eye strain. Neuroscientists have demonstrated striking changes in the caliber of intracranial arteries immediately before and during migrainous episodes. The factors provoking the resultant change in cerebral blood flow, however, remain unidentified.

Migraine therapy, today, provides ways of suppressing and sometimes even aborting the attacks of the disease for most sufferers. But for centuries both patients and physicians were humbled by the ruthlessness of migraine. Therapies certainly abounded in abundance, varying from those which were futile to those which were life threatening. William Harvey, England's most distinguished physician and the man who first described the internal circulation of blood, accepted Viscontess Conway as a patient. Her Ladyship, a renowned scholar, had visited innumerable London physicians to no avail and finally was brought to the great Doctor Harvey. In despair, Harvey recommended that she go to France for a trephination. She wisely refused his suggestion.

A migraine attack is a demoralizing experience. Its pain becomes pervasive depriving its victim of any room to think, to weigh alternatives, to recall joys, to reflect upon past intervals blissfully free of head pain. Dr. Macdonald Critchley, one of England's leading neurologists and a lifelong victim of migraine, once observed: "Migraine is one of those ills when the body has to bear patiently what the soul cannot carry."

– Stanley M. Aronson, MD

Contemporary Management of Rheumatoid Arthritis

Harold Horwitz, MD, and John Conte, MD

Rheumatoid arthritis is an autoimmune, progressive, polyarticular inflammatory arthritis of unknown etiology which affects approximately 1% of the population and costs an estimated 14 billion dollars annually in health care and lost wages. Although archaeological evidence has documented rheumatoid disease in Egyptian mummies and ancient Peruvian Indians, treatment options had been largely symptomatic until well into this century.[1] This review will outline the pathogenesis of rheumatoid arthritis which has provided the rational to replace older empiric therapies with newly conceived therapeutic protocols and bioengineered pharmaceuticals. We will describe the mechanism of action, when known, of current anti-rheumatic compounds, their side effects, and the place each plays in the new therapeutic paradigm.

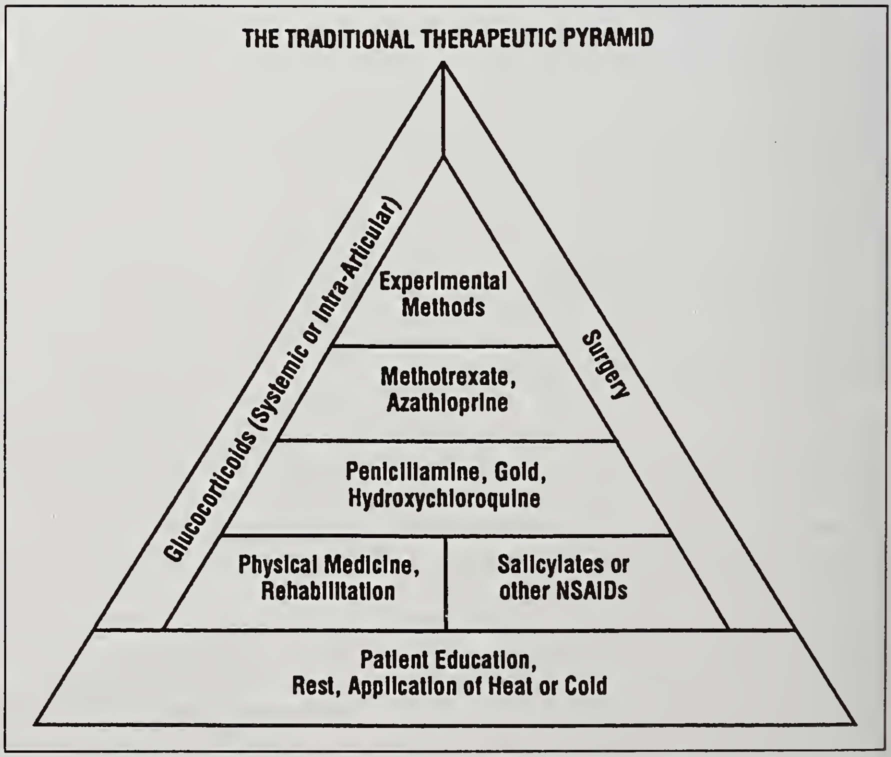
The management of rheumatoid arthritis is as complex now as it was twenty years ago but for different reasons. We have progressed from a situation in which there were few options to offer the patient to one in which choice abounds. For many years the concept of a therapeutic pyramid (Figure 1) dominated the thinking of rheumatologists. This basic program of salicylates (or NSAIDs), rest, physical therapy and patient education formed the keystone of management until the disease progressed to a point of pain and disability that impelled the physician to add gold or hydroxychloroquine, penicillamine or azothiaprine in serial succession in an effort to gain control of the inflammatory process. To the patients suffering during these trial intervals it probably felt as if they had, in fact, built a pyramid! Unfortunately 60% of them would be disabled within 5 years.[1] By the time significant immunosuppressive or disease modifying antirheumatic drugs (DMARD) were added with their attendant toxicity, it was often too little, too late to salvage joint and tendon integrity and restore function.

Abbreviations Used:

COBRA	Combinatietherapie Bij Rheumatoide Artritis
CSA	cyclosporine
DMARD	disease modifying antirheumatic drugs
HCO	hydroxychloroquine
LFT	liver function tests
MR	magnetic resonance
MTX	methotrexate
NSAID	non-steroid anti-inflammatory drug
RA	rheumatoid arthritis
RAIN	Rheumatoid Arthritis Investigational Network
SAS	sulfasalazine
TCR	T cell receptor
THF	tetrahydrofolate
TNF	tumor necrosis factor
UMP	uridine monophosphate

It is well recognized that severe irreversible joint damage usually occurs within two years from the onset of rheumatoid disease.[2] X-ray may not be adequate to assess this but MR studies show these changes readily. Some patients may be in the fortunate 10% who spontaneously remit or have a benign course such that less aggressive therapy will be satisfactory. Certain factors are helpful in selecting those with a poor prognosis such as the presence of rheumatoid factor, a family history of rheumatoid arthritis, systemic symptoms, rheumatoid nodules, an elevated C-reactive protein, and the HLA class II DR4 epitope.

Three developments changed the thinking about therapy. Studies by Pincus[3] showed that the 5 year survival for patients with stage III or IV disease was no better than similarly staged patients with congestive heart failure.[2,3] The addition of methotrexate into the therapeutic armamentarium[4] and the si-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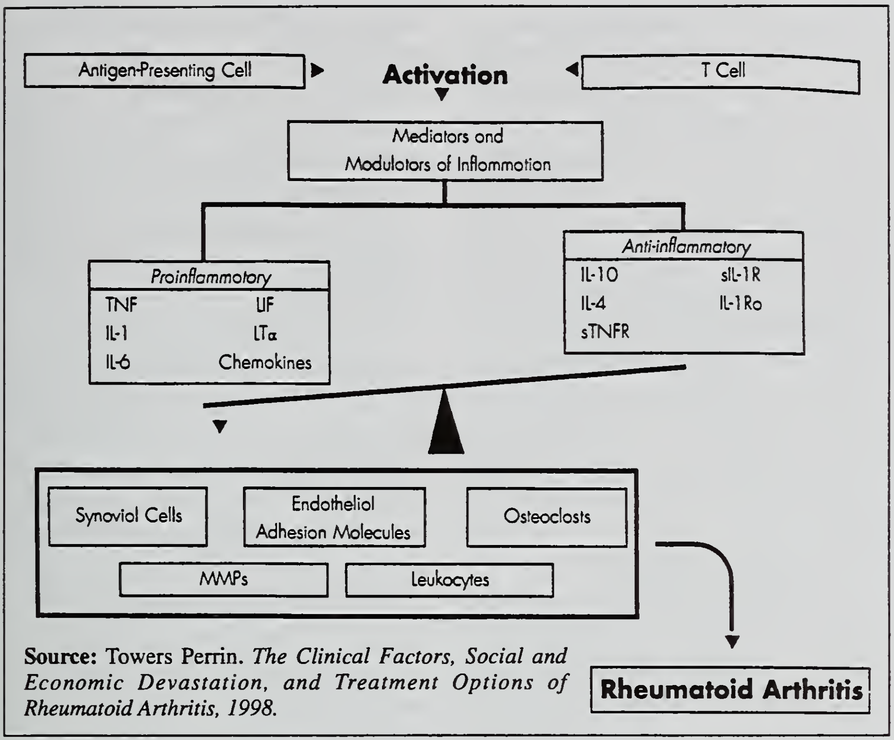
Patient Education, Rest, Application of Heat or Cold

Figure 1.



Source: Towers Perrin. *The Clinical Factors, Social and Economic Devastation, and Treatment Options of Rheumatoid Arthritis, 1998.*

Figure 2.

multaneous application of a combination of DMARDs[5] set the tone for a new era. Also a better understanding of the immunologic cascade of T cells, cytokines and antigen processing laid the foundation for newly-fashioned therapies described in this article.

Currently the pathophysiology of RA (Figure 2)[6] begins with the antigen presenting cell or macrophage recognizing an unknown foreign antigen via the MHC locus and presenting it to the T cell receptor (TCR). Activation of T-cells releases TNF-b, IL-1 and other proinflammatory cytokines which amplify the process in the serum. Within the synovium the macrophage may play a more critical role in which large amounts of TNF-a (4-5x normal) are elaborated. Plasma cells, lymphocytes and fibroblasts congregate to form the destructive rheumatoid pannus. With this as background let us examine the therapies.

We will not dwell on the NSAIDs. (See Habershaw, "Cox-2 Inhibitors," this issue). While they afford symptomatic relief for many patients throughout the course of their disease, NSAIDs are insufficient therapy for the vast majority of RA patients considering what we know about the benefits of DMARDs. Briefly, the toxicity of modern DMARD therapy is rarely as severe as the untreated natural history of rheumatoid arthritis.

Dating back to the 19th century, gold salt was the first DMARD used in RA. Originally employed as a possible antibacterial to combat what was theorized to be an infectious cause of rheumatoid arthritis, it has continued to be used on an empirical basis while its definitive mode of action remains elusive. Administered intramuscularly as either Myochrisine® or Solganol® and also available as Ridura®, an oral but slightly less effective form, it can induce partial remissions in 60% of patients but at a relatively high toxic to therapeutic ratio. Proteinuria, thrombocytopenia, rash, oral ulcerations and other toxicities cause many patients to discontinue the medication within 2 years. However, in those patients who tolerate it, gold can be continued for years. IM gold is dosed at 25 to 50mg on a weekly basis till a response is achieved or a one gram total loading dose is reached. It may be continued weekly or often reduced to biweekly or bimonthly dosing. Long lasting remissions have been observed in a subset of patients, a phenomenon that appears unique to gold compounds.

Originally developed as an anti-malarial, hydroxychloroquine, Plaquenil®, is another drug that has stood the test of time in the management of rheumatoid arthritis, lupus and various dermatologic disorders. Employed since the 1950s, this drug is given orally in a dose of 400-600mg, and generally requires 3-4 months for its full effect to be manifest. It is the safest and best tolerated of the DMARDs, with nausea and rash as the main side-effects. Macular retinopathy is a concern with the cholroquine drugs but the incidence with Plaquenil is very low indeed. It is a time- and dose- dependent toxicity; therefore, monitoring of visual fields and color vision is recommended every 6 -12 months. The mechanism of action includes stabilization of lysosomal vacuoles inhibiting the release of inflammatory enzymes from white cells of the immune system. A recent study by Clark has confirmed its benefit.[7]

Sulfasalazine (Azulfidine®) is a weak folate anatagonist which is a combination of a sulfadine moiety and 5-aminosalicylate and which, like gold, was originally designed as a way to treat the presumed infectious cause of rheumatoid arthritis as well as deliver an anti-inflammatory bypassing the stomach. Unfortunately the amount of salicylate absorbed does not achieve a therapeutic level. The sulfa portion, however, may confer some mild immune suppression. Studies[8] vary as to its effectiveness when it is used alone and in combination with other DMARDs. Azulfadine's toxicity is essentially similar to other sulfa compounds and features fever, thrombocytopenia and rash, including erythema multiformae.

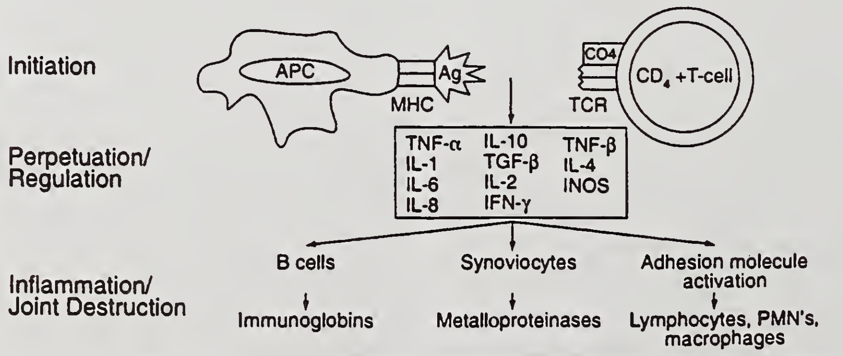
Methotrexate is currently the pre-eminent drug for RA.[8] It is given as a once weekly oral or IM dose usually starting at 7.5 mg and increasing gradually to doses as high a 25mg with most patients responding at doses of 10-15mg. Pretreatment screening for underlying liver disease with viral hepatitis B and C serologies, liver function tests, creatinine and chest x-ray is mandatory. Patients with a history of significant alcohol use, chronic viral hepatitis or other causes of liver disease or dysfunction or renal insufficiency are generally proscribed the use of this drug. In stable patients, LFTs, creatinine and CBC are monitored every 2 months.

One of the challenging issues with methotrexate therapy is whether to perform liver biopsies in the face of normal liver function and LFTs. The American College of Rheumatology has published guidelines for monitoring methotrexate toxicity and, when followed, the probability of missing significant liver disease is low and a biopsy can usually be avoided. Daily supplementation with folic acid may prevent some of the hematologic and GI toxicities. In cases of severe toxicity, folinic acid, Leukovorin, may be utilized. Methotrexate pulmonary toxicity is another serious and unpredictable side-effect that can occur at any time in the course of therapy. Certain risk factors that have been identified are previous rheumatoid lung disease, diabetes, older age and low serum albumen. The patient should not be rechallenged as the second response may be worse.

Methotrexate's mechanism of action is varied in theory and not known with certainty with regards to its anti-rheumatic effect. Tetrahydrofolate (THF) serves as a methyl donor for a whole chain of biochemical reactions which lead to the accumulation of polyamines within the cell. These proinflammatory polyammines are present in excess in RA and over stimulate the immune system. Methotrexate inhibits the formation of THF, making fewer polyamines available. Other mechanisms of antirheumatic action are theorized, including macrophage inhibition. After administration, methotrexate is polyglutamated and remains inside cells for some time. In this active form it can inhibit 5 aminoimidazole-4-carboxamide ribonucleoside transformylase which through a series of steps leads to the accumulation of adenosine in both the intra and extracellular milieu. Adenosine has antiinflammatory effects in a variety of animal and laboratory models of inflammation. Interestingly, sulfasalazine may have a similar mechanism of action via the adenosine pathways.

Combination therapy for rheumatoid arthritis has been studied in two major studies. COBRA (Combinatietherapie Bij Rheumatoide Artritis) from the Netherlands in which sulfasalazine 2gms/d, methotrexate 7.5mg/wk and Prednisone in tapering doses over 6 weeks was compared to SAS alone. After 56 weeks 49% of the combination group versus 27% in the SAS only group had achieved a 50% improvement.[9] A second study in the Netherlands used either MTX or SAS alone or in combination, and again the results favored the combination group.[10] In the United States the RAIN (Rheumatoid Arthritis Investigational Network) combined MTX , SAS and HCQ together or SAS and HCQ, or MTX alone. Patients who had previously failed one or more DMARDs were eligible. SAS was used at a fairly low dose of 500 mg BID. After 2 years 77% of the triple therapy group, 40% of the MTX group and 33% of the SAS-HCQ group had improved. The treatment failures were then put into the triple therapy group, and, after three years 75% of these were also at least 50% improved.

The Inflammatory Cascade in Rheumatoid Arthritis



Major cells and cellular products implicated in the immune-mediated inflammatory process of rheumatoid arthritis. APC=antigen-presenting cell; Ag=antigen; MHC=major histocompatibility complex; TCR=T-cell receptor; TNF-α=tumor necrosis factor α; IL-l=interleuken-1; TGF-β=transforming growth factor β; INOS=inducible nitric oxide sythase; PMN's=polymorphonuclear

Adapted from Moreland et al. *Arthritis Rheum* 1997; 40:398

Source: Frank Graziano. "Remodeling the Treatment Pyramid in RA," *Rheumatoid Arthritis Management Today,* 1999; 1:3.

Figure 3.

Cyclosporin (Neoral® or Sandimmune®) has been approved by the FDA for use in combination with MTX for severe rheumatoid arthritis patients who are partial responders to MTX alone. The results from Tugwell's Canadian study demonstrated that the combination was supperior to either alone and, importantly, allowed a lower, less toxic dose of CSA to be employed. In this study of only 6 months duration with a open label 6 month extension, MTX plus CSA was compared to MTX alone. During the first 6 months the combination group did approximately 25% better than the MTX alone group. During the extension period the new combination group equalled the responses of the original group.[11]

The mechanism of action of CSA may be the inhibition of IL-2 synthesis by CD-4 lymphocytes. IL-2 is essential for the clonal activation and expansion of T-cells. CSA also inhibits expression of the CD40 ligand on activated T cells which is necessary for proliferation of B cells.

The dosage of CSA must be carefully titrated to avoid renal toxicity. In RA a dose of 2.5-4.0 mg/kg seems appropriate. If the creatinine rises 30% above baseline or hypertension ensues then the medication must be either reduced or stopped. Peripheral calcium channel blockers are used to treat secondary hypertension. Caution should be exercised in patients with liver or kidney disease, concomitant NSAIDS therapy or drugs which inhibit or are metabolized by the p450 system. The dosage is initiated at 2.5 mg/kg and titrated by 0.5 mg/kg increments per month. If a satisfactory response is not achieved by 6 months then CSA should be discontinued. In early monotherapy studies for RA utilizing a high dose of CSA, patients had a 60% response rate but with unacceptable renal toxicity.

Leflunomide (Arava®), approved for use November 1998, is an inhibitor of the enzyme diihydroorotate dehydrogenase, an enzyme critical for the formation of uridine monophosphate. As activated lymphocytes pass through the G1 phase, lowered levels of UMP arrests cellular replication. Resting T cells are able to maintain metabolism by salvage pathways and remain viable. In a variety of models leflunomide has been able to reduce the immune response. In addition to its action on autoimmune lymphocytes this inhibitor of pyrimidine synthesis has been shown to inhibit leukocyte adhesion to vascular walls and reduce the production of the immunostimulatory cytokine IL-2. Leflunomide also exhibits antiinflammatory effects on cyclooxygenase II and to a lesser extent on nitrous oxide synthetase.

In clinical studies leflunomide performs as well as methotrexate achieving a 75% ACR 20 % response rate.[12,13] The drug is administered with a three-day loading dose of 100mg, followed by daily doses of 10 to 20mg. Leflunomide is actually a pro-drug and is rapidly and non-enzymatically converted to its active form, a malononitrilamide and recirculated through the enterohepatic circulation. The major toxicities are a reversible elevation of AST and ALT, alopecia and diarrhea. An increased rate of infections has not been reported. About 17% of patients experience side effects which necessitate the discontinuation of the medication or treatment with cholestyramine which quickly eliminates it within a matter of a few days. Monitoring consists of monthly liver function test and blood counts. This drug offers a viable alternative to methotrexate and the other DMARDs and can also be used in combination with MTX for partial responders.

Etanercept (Enbrel®), the first bioengineered drug approved for rheumatoid arthritis, is a tumor necrosis factor (TNF) receptor fusion protein which is given subcutaneously twice a week . The drug binds and neutralizes circulating TNF and reduces its availability to stimulate the immuno-inflammatory cascade. Within approximately 3 months 60% of patients achieved an ACR response of 50% and 75% achieved a 20% response.[14] Weinblatt recently demonstrated an enhanced and prolonged effect when Enbrel was combined with methotrexate.[15] As with MTX, Enbrel therapy must be continued for clinical benefit to be maintained. In prerelease clinical trials no significant side effects were noted with Enbrel other than local skin redness and puritus at the injection site. No specific laboratory monitoring is currently recommended. Concerns, however, over the possibility of increased risk of sepsis were raised when recent case reports surfaced documenting respiratory infections, septic arthritis and bacteremias. These have usually involved patients with poorly controlled diabetes, open skin ulcers, and advanced debilitating rheumatoid arthritis in whom the risk of infection has historically been greater. The manufacturer has added an advisory statement as an addedum to the drug labelling cautioning clinicians to be aware of this possiblity. As with any new promising agent the passage of time and more generalized use will better define its risk-to-benefit ratio.

> *The management of rheumatoid arthritis is as complex now as it was twenty years ago but for different reasons. We have progressed from a situation in which there were few options . . . to one in which choice abounds.*

Another means of neutralizing the effects of TNF is by using a monoclonal antibody which binds both circulating and cell bound TNF. This medication (infliximab®) has been approved for use in Crohn's disease and has shown promise in RA. Dosing studies are now underway. Again it appears that MTX is helpful in sustaining response by inhibiting the formation of neutralizing antibodies.

Minocycline and the use of other antibiotics in RA is "old wine in new bottles." Theories of the etiology of RA have always included infection - whether bacterial, viral and even protozoan. The finding of infectious etiologies for certain rheumatoid factor negative inflammatory arthritides such as Reiter's syndrome, Lyme disease and Parvovirus gives us pause to consider that an infection may still be at the root of most rheumatoid disease. In the 1950s Dr. Lorand Brown was a vocal advocate of the use of tetracycllines in management of many types of arthritis. His views were not generally accepted since careful controls were never done nor presented in a peer-reviewed journal. Subsequently, three double blind studies were done and demonstrated efficacy. These studies include a study by Kloppenburg[16], the MIRA study by Tilley[17] and by O'Dell.[18] These studies can be summarized as showing a response rate of 38-65% compared to placebo improvement rates of half that amount. The mechanism of action may be several fold and includes the inhibition of the metalloproteinase enzymes like collagenase, reduction in leukotaxis, decrease in production of phospholipase A2, and a decrease in T cell proliferation after CD3 antibody stimulation. Most researchers currently believe the anti-rheumatic benefits of the tetracyclines are mediated through these cellular effects that are unrelated to their antibiotic actions.

The toxicity of tetracycline includes the 8th nerve, causing dizziness and vertigo, a bluish discoloration of the skin, phototoxicity, nausea, and occasional vaginitis. There is no significant increase in infections at the usual dose of 100mg BID. It remains to be seen whether these initial successes can continue to be replicated. At the very least, it has stimulated further research with similar compounds.

What role does prednisone, an inhibitor of Lipomodulin, and other corticosteroids play in RA? When initially developed in the 1940s at the Mayo Clinic these powerful anti-inflammatory compounds were thought to be the "magic bullet" for RA. Unfortunately serious side effects at high doses soon became obvious. However low dose prednisone (less than 7.5) clearly has salutory effects on the rate of joint destruction with far less toxicity.[19] When one or two joints flare out of proportion to the overall trend of the disease and infection is ruled out, then local corticosteroid injection can be quite beneficial and safe. Oral treatment is commonly initiated early on in RA to rapidly suppress inflammation till longer acting DMARDs can take effect.

The length of this review does not allow us to discuss azathioprine, thalidomide, the omega-3 fatty acids among other older drugs and some of the emerging therapies such as anti-CD 4, anti IL-1, IL-10 enhancement to mention just a few.

How this long laundry list of treatment options is assembled into a "well-dressed" patient requires the skill of the rheumatologist, the cooperation of a well-informed patient, the help of the support programs of the Arthritis Foundation and physical and occupational therapy.

In summary, pharmacologically one still generally begins but rarely ends with an NSAID. The new selective COX-2 inhibitors may eventually become the first line NSAID but more documentation of their efficacy in RA will be required. Occasionally low dose steroids may have a better risk - benefit ratio, especially in the older seronegative patient. At the same time a DMARD or combination of DMARDs should be started. New biologics such as Enbrel could replace MTX as the first line DMARDs in moderately severe patients because of increased efficacy if concerns about infections and cancer risk are allayed as longer term experienced is gained. Leflunomide is a logical choice now over methotrexate in patients with concomitant pulmonary disease, nodulosis and in some patients with a risk of chronic liver disease. Methotrexate will almost certainly continue to be used in combination with both Enbrel and Leflunomide in patients with aggressive disease and as single agent in early to moderate RA. Often initiating hydroxychloroquine or sulfasalazine early on in the treatment for rheumatoid arthritis is valid for many patients because of their favorable side-effect profiles and the possibility they may facilitate a quicker taper of potentially more toxic drugs. Minocycline or similar metalloproteinase inhibitors could become commonplace in rheumatoid protocols to protect cartilage from destruction that escapes previously initiated therapies?

As we look to the future our expanding ability to selectively manipulate the immune and inflammatory response in patients with rheumatologic disease will continue to improve. We are closer now to controlling the ravages of rheumatoid arthritis and allowing our patients to enjoy lives that avoid the pain, disfigurement and surgeries that all too often accompany this most aggressive of arthritis conditions.

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Update on Osteoarthritis

Stuart T. Schwartz, MD, and Bernard Zimmermann, MD

Sixteen million Americans suffer from osteoarthritis (OA), the most common rheumatic disease. OA is characterized by loss of the articular cartilage, sclerosis and eburnation of the underlying bone, ostophyte formation and subchondral cysts. Clinical features include: joint pain, tenderness, limitation of movement, crepitus, joint effusion, and at times local joint inflammation. More than 80% of individuals over the age of 55 have radiographic evidence of OA but x-ray findings do not necessarily correlate with symptomatic disease;[1] 10-30% of those affected will have significant pain or disability.[1] The impact of OA to society is enormous: nearly 73 million physician visits and 36 million days lost from work annually.[2]

Etiology and Pathogenesis

Trauma and repetitive stress have been implicated as risk factors in the pathogenesis of OA. In occupations such as mining and weaving, a clear relationship exists between the joints used during work and the development of OA. This correlation has not been demonstrated with recreational exercise such as long distance running in people with normal joints.[3] Obesity predisposes to OA of the knee but not of the hip in women.[4] Weight loss of 5 kg was associated with a 50% reduction in the development of symptomatic OA of the knee.[5] OA of the knee occurs more commonly in African American women than in Caucasians. The prevalence of OA increases with age.

In 1941 Stechner recognized the importance of genetics and gender in OA, when he found that Heberdens nodes (bone and cartilage enlargement of DIP joints) were inherited as an autosomal dominant trait in women, but as an autosomal recessive pattern in men.[6] Heberdens nodes occur approximately 10 times more often in women than men. Women whose mothers or sisters have Heberdens nodes have a 2 - 3 times greater risk of nodal OA. The pattern of joint involvement is similar in young adults but in women over 55 years old, hand joints are more commonly affected.[7] The hip joint is affected more often in men than women.[7] The genetic link in families with premature generalized OA was found in 1990 with the discovery of a mutation in the type II collagen gene (Col2A1).[8] A single amino acid substitution at position 519 converting arginine to cysteine results in precocious generalized OA and mild chondrodysplasia.[9] Premature OA and gene substitutions in types II, IX, and XI collagen have now been found in other families.

Abbreviations Used:

ACR	American College of Rheumatology
AMA	American Medical Association
CS	chondroitin sulfate
FDA	Food and Drug Administration
GS	glucosamine sulfate
HA	hyaluronic acid
COX	cycloxygenas
NSAIDS	non-steroid anti-inflammatory drugs
OA	osteoarthritis
TENS	transcutaneous electrical nerve

Recent studies show other associations with OA. The Framingham study notes that women on estrogen replacement therapy have a lower prevalence of OA of the knee and hip.[10] This study also revealed an increased progression of OA of the knee when afflicted individuals had low dietary vitamin D intake and low serum levels of vitamin D.[11] However, the initial development of OA was not related to vitamin D levels. Weakness of the quadriceps muscle is felt to be a risk factor in the development of OA of the knee. Knee extensor weakness may predispose one to develop pain, disability and progression of OA. Strengthening the quadriceps can decrease pain and improve functioning.[12]

Signs, Symptoms and Diagnosis

Osteoarthritis commonly presents with pain and stiffness in older adults. The joints most frequently involved include: knees, hips, feet, distal and proximal finger joints, the base of the thumb (first carpometacarpal joints) and the cervical and lumbar spine. Typically patients have morning stiffness lasting only a few minutes, which improves with activity. Periods of inactivity, such as sitting, cause transient stiffness known as the gel phenomenon. Joints swell because of an effusion or bony hypertrophy. Affected patients report impairment in gait due to pain, instability or loss of movement. When the knee is involved, patients describe a grinding or cracking sensation when walking. Late manifestations of osteoarthritis include nocturnal pain and work disability.

On physical examination, limited range of motion caused by osteophyte formation and non-inflammatory joint effusions may be detected. Crepitus (caused by irregularity of the articular surface) can often be palpated or may even be audible. Frequently, malalignment of the joints can occur, especially in the knee. Various knee deformities result from asymmetric loss of the medical compartment causing a bow legged stance. Signs of local inflammation such as redness and warmth occur less frequently in osteoarthritis compared to inflammatory arthritides (i.e., rheumatoid arthritis or crystal disease). There is however a variant of osteoarthritis, erosive osteoarthritis, which involves the interphalangeal joints of the hands and

Table 1

NONPHARMACOLOGIC THERAPY FOR OSTEOARTHRITIS

- Patient education
  - Weight loss
  - Physical therapy and occupational therapy
    - Range of motion exercises
    - Strengthening exercises
    - Assistive devices
    - Joint protection instruction
  - Aerobic exercise program

(Modified from Hochberg MC, Altman RD, Brandt KD, et al. [13,14])

presents with significant inflammation and radiographic erosions.

The diagnosis of osteoarthritis is primarily based on the characteristic clinical history and objective physical findings described above. Laboratory testing adds little to the diagnostic evaluation since blood tests are usually normal or reflect some other pathologic process. Arthrocentesis typically reveals non-inflammatory effusions with clear or straw colored viscous synovial fluid with a white blood cell count less than 2000. Radiographs usually reveal joint space narrowing, subchondral bony sclerosis, subchondral cyst formation, and osteophytes. Remodeling of the joint surface and changes in bone shape can alter the contour and shape of the bone.

THERAPY

Patients and physicians alike often fail to recognize the many effective modalities of therapy. Patients frequently ask for the newest prescription medication and physicians are often willing to prescribe it without considering alternatives or adjunctive non-pharmacologic therapy. A comprehensive plan for maximum relief of pain and maximizing function in patients with OA should at least include physical measures such as regular strengthening and range of motion exercise, the use of analgesics, and possibly topical modalities and assistive devices

Table 2

PHARMACOLOGIC THERAPY FOR OSTEOARTHRITIS

- Topical agents
- NSAIDS
- Analgesics
  - Acetaminophen
  - Narcotic analgesics
- Intraarticular injection
  - Corticosteroids
  - Hyaluronic acid

The American College of Rheumatology (ACR) and the American Medical Association (AMA) guidelines for the management of osteoarthritis of the knee and hip[13,14] emphasize the role of non-pharmacologic therapy in treating these disorders (Table I). Specific recommendations include patient education, which ideally is accomplished in a formal educational program such as those offered by the Arthritis Foundation, weight loss in the obese patient, and physical and occupational therapy for range of motion exercises and evaluation for assistive devices and adaptive equipment.

In addition to the known cardiovascular and psychological benefits of exercise, an appropriate exercise program will help reduce pain and improve functional capacity in patients with OA. Formal studies have shown that isometric strengthening of the quadriceps and hamstring muscles is superior to passive range of motion exercise in reducing pain and improving function in OA of the knee. A strengthening program may be followed by non-weight bearing aerobic exercise (such as a pool or bicycle exercise program), which will facilitate weight loss and improve cardiovascular and musculoskeletal exercise tolerance.

Patients with more advanced OA of the lower extremity may benefit from canes, braces, walkers and other devices. The use of a cane in the contralateral hand may reduce joint reactive forces in the hip by as much as 50%. The importance of the role of the physical and occupational therapists in this aspect of treatment can not be overemphasized. These professionals have the expertise to evaluate the patients' needs and recommend appropriate equipment, as well as to help the patient overcome psychological barriers, which often hinder progress.

Other physical modalities include thermal therapy (either hot or cold) as an adjunct to exercise. Transcutaneous electrical nerve stimulation (TENS) has been used to treat various types of pain, but there is insufficient data to support its use for the treatment of OA. Similarly, acupuncture has a transient pain relieving effect but studies have not shown any durable benefit for patients with OA.

The non-steroid anti-inflammatory drugs (NSAIDs) have been widely used as initial therapy. However, the increased appreciation for the serious side effect potential of these medications has led to a shift toward the use of non-NSAID analgesics and intensified research efforts aimed at the development of less toxic anti-inflammatory medications. The 1995 ACR guidelines for the treatment of OA of the knee and hip recommend acetaminophen at does up to 1 gram qid as initial pharmacologic therapy.[13,14] (Table 2). If there is synovial effusion in an osteoarthritic knee, aspiration and injection of corticosteroids should be considered. Topical capsaicin should also be tried before the NSAIDs. As gastrointestinal bleeding has been shown to be related to the dose of NSAID, low-dose NSAID is recommended. If all of these measures fail to produce an adequate improvement in a patient's symptoms and functional capacity, then full-dose NSAID may be used. The patient with risk factors for upper gastrointestinal bleeding or history of ulcer disease should receive misoprostol or another gastroprotective agent.

There have not been clinically significant differences in efficacy shown among the various NSAIDs. Individual patients will have variable response to various NSAIDs, which cannot be predicted in advance. Thus, the clinician's choice of prescription should be based on cost, convenience and side effects. A short-acting, inexpensive NSAID, such as ibuprofen

400-800 mg every 6-8 hours may be best for a patient with intermittent ambulatory hip pain, while a patient with OA of the hands who is working all day may find the added cost of a one-a-day preparation (e.g., oxaprocin 1200 mg. every AM) worthwhile. Some of the currently available NSAIDs vary in their adverse effect potential. Indomethacin, for example, probably has a higher incidence of gastrointestinal and central nervous system toxicity than other NSAIDs and should be reserved for short-term use. The non-acetylated aspirin products such as salsalate and chlorine magnesium trisalicylate have been associated with a low incidence of gastrointestinal toxicity and bleeding but are limited by twice-a-day dosing and large pill sizes.

Progress in the search for less toxic anti-inflammatory drugs has been accelerated by the discovery two isoforms of cycloxygenase (COX). It is now known that one form of COX is responsible for inflammation (COX-2) and the other (COX-1) is required for homeostasis in the stomach and kidneys. This advance has led to the development of agents that selectively inhibit COX-2 in hopes that an anti-inflammatory and analgesic effect can be achieved with a reduced potential for gastrointestinal ulceration and bleeding. (See Thomas Habershaw, "Cox-2 Inhibitors," this issue.)

In 1997 the FDA approved viscosupplementation with intraarticular hyaluronic acid (HA). HA is a normal constituent of synovial fluid, composed of repeating units of glucuronate and N-acetylglucosamine. HA is also a component of articular cartilage in which it functions by aggregating proteoglycans into large macromolecular units, which confer elasticity and resistance to compression of the cartilage. Synovial fluid in individuals with OA has a lower concentration of HA, which is felt to reduce the natural protection from mechanical stress.[15] Intraarticular injection of HA has been shown to decrease pain in knees affected by OA.[16,17] The mechanism by which HA works is not fully understood, but it does not act solely as a lubricant. Although injected HA is only present in the joint for a few days, the resulting pain relief can last 6 to 12 months. Experimental *in vitro* studies have shown that injection of HA stimulates synovial-lining cells to produce endogenous HA and activate chondrocytes to synthesize proteoglycans.[18] HA is felt to have an effect on intraarticular pain receptors.[15] HA can inhibit macrophage migration, phagocytosis, release of prostaglandins, and impair the migration of granulocytes.[13] It may also function to clear debris and inflammatory products from the joint. Whatever its mechanism of action, HA given as a series of 3 to 5 weekly intraarticular injections has been shown to be equivalent to 26 weeks of acetaminophen or naproxen.[16,17] Infrequent side effects may include local irritation at the injection site, headache and gastrointestinal complaints. Hyaluronic acid is derived from the combs of rooster. Significant allergic reactions are rare but administration to individuals with allergies to eggs or poultry should be initiated with caution. There is no evidence at this time that HA alters the natural history of OA or prevents the progression of cartilage loss.

Patients and physicians alike often fail to recognize the many effective modalities of therapy.

Glucosamine sulfate (GS) and chondroitin sulfate (CS) have been claimed by some to "cure" arthritis.[20] While these substances are normally present in the connective tissue matrix of cartilage, their effectiveness when given as oral supplements is unproven. Most of the clinical studies evaluating the role of GS and CS in treating OA were conducted in Europe. Small numbers of patients who were followed over 4 - 8 weeks showed a modest benefit in controlling symptoms of OA, (i.e., pain, tenderness and swelling) equivalent to low-dose ibuprofen.[21] Fortunately, the side effect profile of GS and CS appears to be relatively benign with mild gastrointestinal upset as the most common adverse reaction. Long term studies following large numbers of patients are needed to evaluate the safety and effectiveness of these food supplements. There is no evidence at this time that oral ingestion of GS and CS will result in their integration into new connective tissue matrix.[21] There also is no convincing evidence of long-term protective effects of GS and CS on cartilage. The purity and content of each tablet cannot be assured since they are nutritional supplements and not regulated by the FDA. Other unproven remedies are listed in Table 3.

Table 3

UNPROVEN REMEDIES COMMONLY USED FOR OSTEOARTHRITIS

- Magnet therapy
- Copper bracelets
- Glucosamine sulfate
- Chondroitin sulfate
- Ginger extracts
- Shark cartilage
- Gin-soaked raisins

Future therapies for OA will attempt not only to control symptoms but to alter the rate of progression of the disease. Both surgical and pharmacologic modalities are currently in various stages of research and development. The term "structure modifying" agent has been recommended to indicate drugs that are intended to prevent, retard, stabilize or revise the development of OA. This terminology emphasizes OA as a disease of the entire joint, not just a disease of articular cartilage.

Two promising agents with structure-modifying potential are the tetracycline family of antibiotics and the metalloproteinase inhibitors. Tetracyclines are inhibitors of tissue metalloproteinase which is thought to be important in the development of OA. Minocycline has been shown to be as beneficial for therapy of rheumatoid arthritis, and studies are underway in OA. Doxycycline inhibits articular collagenase activity and has reduced the severity of OA in canine models. Non-antibiotic inhibitors of metalloproteinase are currently being studied in animals and humans. Other theoretical approaches to modifying the cause of OA under investigation include genetic manipulation of tissue inhibitors of metalloproteinases, cytokines and growth factor manipulation and the use of glycosaminoglycan complexes with polysulfaric acid and peptides.[22]

Surgical approaches to modifying and repairing structural defects in osteoarthritis are also under investigation. Articular cartilage grafts and transplantation using autologous chondral and periosteal grafts have shown promising results for repair of focal cartilage defects.[23] These techniques have been most successful in young patients with small localized cartilage defects, but further refinements may help patients with generalized OA.

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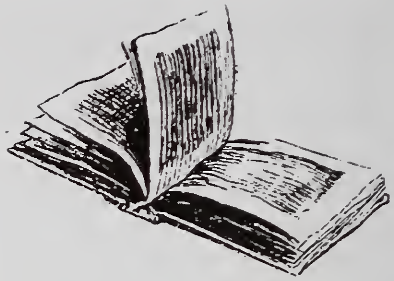

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The Clinical Management of Fibromyalgia

John R. Parziale, MD

EPIDEMIOLOGY

Fibromyalgia is a common disorder causing chronic muscular pain and fatigue which affects affects approximately three million individuals in the United States. Of those affected, 80% are women between 20 to 50 years of age.[1] First described by Balfour in 1824, fibromyalgia is characterized by tender points and trigger points in multiple areas, usually along large muscles or muscle-tendon insertions; Stockman (1904) reported muscular nodules which are "inflamed, tender, and if large, can be easily felt". This disorder has become known by many names including fibrositis, muscular rheumatism and tension myalgia.

The predominant symptom in the vast majority of patients is widespread musculoskeletal pain. Myalgias, polyarthralgias and pain in the neck, mid- and lower back are reported. The pain is often described as sharp, aching, burning or cramping, and may be more severe in one or more locations or occur in "waves" of generalized musculoskeletal discomfort. An important characteristic of fibromyalgia is its fluctuating course; while some degree of pain is persistent, there is a cyclic pattern of "good days" and "bad days".

DIAGNOSIS

Pain is often aggravated by cold or damp weather, poor sleep, intercurrent illness, lack of exercise, trauma, and physical or psychological stress. Ameliorating factors include warm/dry weather, restful sleep, heat application and low-impact aerobic exercise. (Table 2) Many patients have associated complaints of headaches, subjective swelling of the hands or feet, insomnia and irritable bowel symptoms including abdominal pain, cramping or loose stools.

The American College of Rheumatology established diagnostic criteria for fibromyalgia in 1990, with a predicted sensitivity of 88% and a specificity of 81%.[2] Criteria include:

Table 1.
American College of Rheumatology 1990 Diagnostic Criteria for Fibromyalgia

1. Chronic muscular pain of at least 3 months duration.
2. Pain in 11 or more tender points within 18 characteristic locations:
   - Occiput
   - Lower Cervical
   - Trapezius
   - Supraspinatus
   - Second Rib
   - Medial Knee
   - Lateral Epicondyle (Elbow)
   - Upper Gluteal Border
   - Greater Trochanter (Later Thigh)

Abbreviations Used:

ACR	American College of Rheumatology
ATP	adenosine triphosphate
COX-2	cyclo-oxygenase 2
LTR	longitudinal twitch response
NSAID	non-steroid anti-inflammatory drug
REM	rapid eye movement
5-HTP	5-hydroxy-L-tryptophan

1. History of widespread muscular pain in both left and right sides of the body, pain above and below the waist, and pain in the neck, chest, or low back.

2. Pain in eleven of eighteen tender point sites on digital palpation (occiput, lower cervical, trapezius, supraspinatus, second rib, lateral epicondyle, greater trochanter, medial knee). (Table 1 and Figure 1)

A variety of medical conditions including hypothyroidism, rheumatoid arthritis, systemic lupus erythematosus, and myopathic or neurologic conditions should be considered within the differential diagnostic possibilities for chronic muscular pain. (Table 3) It is obligatory to rule out such disorders with a careful history and physical examination as well as the judicious use of laboratory studies.[3]

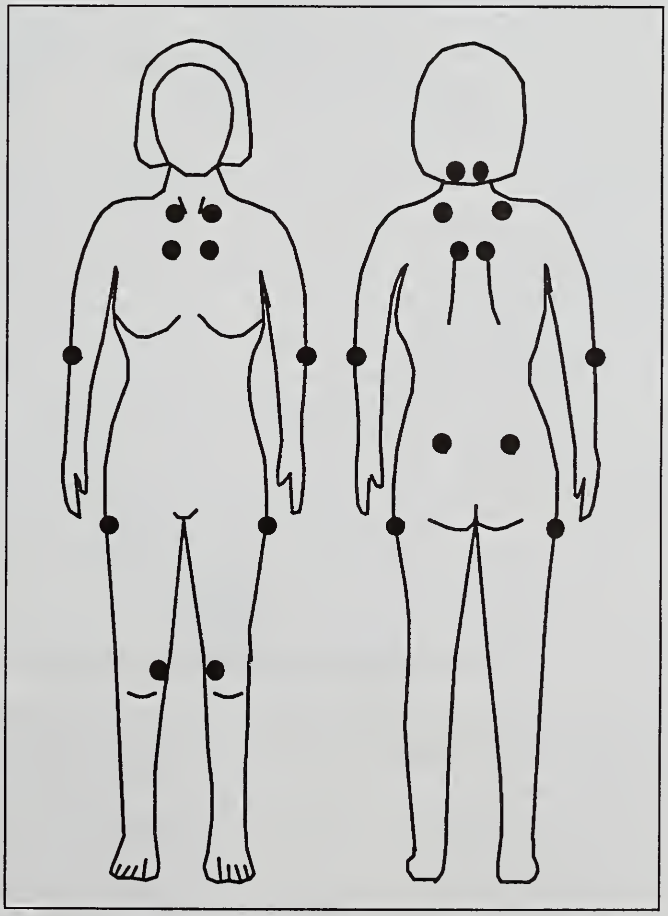

Figure 1: Tender Points Recommended for Diagnosis of Fibromyalgia

PATHOPHYSIOLOGY

The etiology of fibromyalgia is uncertain, and a variety of theories have been developed to explain its pathogenesis.; e.g, abnormalities of the sleep cycle, disorders of muscle metabolism, disturbances of neurotransmitters, alterations of the hypothalamic-pituitary axis, and psychopathologic mechanisms. (Figure 2)

An important physiologic characteristic of many fibromyalgia patients is disrupted sleep, with frequent interruptions and/or terminal insomnia. Normal sleep involves cycles of four non-dream stages known as Non-REM sleep followed by a dream stage called REM (rapid eye movement) sleep; these five-stage cycles repeat every 45 to 60 minutes. Moldofsky described alpha-wave intrusion of delta sleep in Stage IV.[4] In Stage IV, EEG activity represents predominantly delta waves, indicating deepest sleep. Delta waves are presumed to be necessary for restoring muscles histochemically. Deprivation of sleep in healthy persons, in particular those with a low fitness level, may elicit transient symptoms similar to fibromyalgia.[5]

CLINICAL MANAGEMENT STRATEGIES

The first step in managing the individual with fibromyalgia is making an accurate diagnosis.[6] Many patients have seen multiple physicians, without a clear diagnosis for their constellation of symptoms. A careful explanation to the patient of the concept of fibromyalgia is important. Patients should be informed that the prognosis for improvement in fibromyalgia is generally good, but that some pain may be chronic. Educational material is frequently helpful, and is available through organizations such as the Arthritis Foundation. Patients may gravitate toward a "fibromyalgia support group", which can be a mixed blessing: while some report a sense of relief in receiving medical updates and in knowing they are not alone in their disease, others indicate that group meetings disseminate misinformation or deteriorate into "whining sessions". The Internet has offered a new vehicle for information sharing, though the information distributed is not always factual. A reasonable alternative may be *Arthitis Today* or the *Fibromyalgia Wellness Letter*, publications of the Arthritis Foundation with a medical advisory board which includes several nationally recognized experts in rheumatology and related fields.

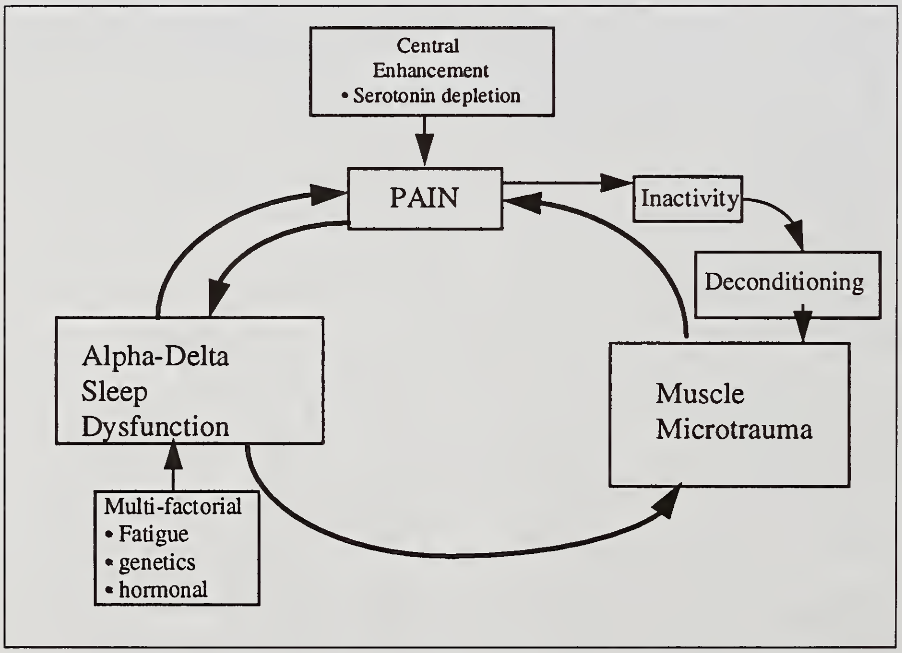

Figure 2. Pathophysiology of Fibromyalgia

Table 2.
Factors Affecting Fibromyalgia Symptoms

Factors Aggravating Fibromyalgia	Factors Ameliorating Fibromyalgia
• Cold or Damp Weather	• Warm or Dry Weather
• Poor Sleep	• Restful Sleep
• Intercurrent Illness	• Application of Heat to Affected Muscles
• Lack of Exercise	• Modest Exercise
• Trauma	
• Physical of Psychological Stress	

A multifaceted approach is often successful (Table 4) and includes:

1) Diagnosis and re-assurance
2) Avoid aggravating factors
3) Medications
4) Exercise

Aggravating factors for fibromyalgia include cold or damp weather, impaired sleep, intercurrent illness, lack of exercise, trauma, and physical or psychological stress. Cold exposure results in muscular contraction or shivering, in an effort to maintain core body temperature for normal homeostasis. The relationship of damp weather to increased symptoms is less clear, though declines in ambient barometric pressure have been associated with arthralgia.

Sleep impairments can be exogenous (noise, vibration, excessive work hours, etc.) or endogenous. In addition to the previously noted alterations in delta sleep, pharmacologic interventions can worsen sleep; caffeine, nicotine or pseudoephedrine may inhibit initiation of Stage I, and benzodiazepines can prolong Stage II or III at the expense of Stage IV. Alcohol and some anti-hypertensive medications cause diuresis.

A heavy meal can cause gastric distention or worsen reflux symptoms; nasal polyps may obstruct the airway or worsen sleep apnea. Melatonin supplements to improve sleeping patterns have little role in fibromyalgia treatment; a recent study found that pre-menopausal women actually had elevated levels of melatonin, while cortisol levels were comparable to age-matched controls.

Waylonis described various occupational activities that are associated with increased pain symptoms.[7] Individuals with fibromyalgia have difficulty tolerating jobs involving overhead assembly or frequent reaching to high shelves; frequent or prolonged computer use, key entry or typing can also pose problems. Workplace

Table 3.
Differential Diagnosis of Chronic Muscular Pain

- Fibromyalgia
- Myofascial pain syndrome
- Arthritis (Rheumatoid or Osteoarthritis)
- Chronic Fatigue Syndrome
- Rheumatic Diseases (Systemic Lupus Erythematosus, Sjogrens Syndrome, Ankylosing Spondylitis)
- Myopathies (Polymyotositis, Dermatomyositis, Myasthenia Gravis, Eaton-Lambert Syndrome)
- Disc Herniation
- Polymyalgia Rheumatica
- Other (Sleep apnea, Reactive Hypoglycemia, Hypothyroidism, Anemia, Diabetes Mellitus Type II, Malignancy)

modification is often beneficial. Working nights may further aggravate sleep disturbance, and excessive visual and auditory stimulation have been observed to aggravate fibromyalgia symptoms.

Inactivity leads to deconditioning and muscle fiber atrophy, as well as joint capsule and tendon contracture. Direct muscle trauma causes swelling and localized pain, but does it actually cause the fibromyalgia syndrome? Though experts may reasonably question the concept of "secondary fibromyalgia" due to accident or injury, there is consensus that physical trauma can worsen an existing fibromyalgia condition. Psychological stressors are also associated with pain complaints, impaired sleep and irritable bowel symptoms.

The single most important strategy in treatment is to interrupt the cycle of pain and fatigue. In general, the longer the duration of pain, the more difficult it is to interrupt the cycle. An essential initial step is to regulate and improve the disrupted sleep pattern. Treatment with cyclobenzaprine or amitriptyline has been shown to increase Stage IV sleep and to decrease myalgias and tender points.[8] Patients using a combination of two antidepressant drugs, amitriptyline and fluoxetine, experienced twice the improvement in pain levels, sleep disturbances and overall functioning as compared with those taking a single medication.[9] Medications such as sertraline and paroxetine are being investigated.

> *Fibromyalgia is characterized by chronic diffuse muscular aches, fatigue and poor sleep; it affects nearly three million individuals in the United States alone, predominantly younger women.*

Non-steroidal anti-inflammatory drugs are helpful in alleviating local pain symptoms and muscle tenderness, but provide little to no sleep regulation effect and may cause gastrointestinal symptoms via inhibition of prostaglandin synthesis. The recently released cyclo-oxygenase 2 (COX-2) agents (see Habershaw, this issue), should minimize adverse side-effects. Acetaminophen and tramadol, two non-narcotic analgesic agents, can be helpful. Investigational field trials using mexilitine, which reduces membrane depolarization by blocking ion transport channels, have shown promise. In a small group of fibromyalgia patients with severe muscle tenderness refractory to first line medications, 50% have reported improved function and reduced pain levels after mexilitine therapy (unpublished). Narcotic analgesics and oral steroids should not be used for chronic pain control in fibromyalgia patients.

Malic acid and magnesium are necessary for the generation of adenosine triphosphate (ATP), a substance thought to be deficient at fibromyalgia trigger points; however, randomized, double-blind studies have not proven any benefit when these substances were taken orally. The protein 5-hydroxy-L-tryptophan (5-HTP) is partially converted to serotonin, a hormone important in the regulation of mood, pain response and sleep. Dietary supplements of 5-HTP have not been proven effective, and may carry risks; the FDA banned the sale of L-tryptophan in 1989 after an outbreak of eosinophilia myalgia syndrome. Guaifenesin, an expectorant found in many cough syrups and touted on the Internet as a fibromyalgia cure, has no proven benefit.

Physical modalities including superficial heat or ultrasound, massage, topical anesthetic sprays, and biofeedback have all been reported as helpful, though controlled studies are minimal. Trigger point injections, with or without injectable steroids, are most helpful if a visible muscle contraction known as a longitudinal twitch response (LTR) is evident. Random injection of diffuse myalgias without an associated LTR is often ineffective. Alternative medicine treatments including acupuncture are used by many fibromyalgia patients - 91% of 80 patients in one study - but patient satisfaction rates were modest for the most commonly used interventions.

Recreational aerobic exercise including cycling or fast-walking has been shown to improve pain levels and reduce fatigue, though sleep was not improved.

Table 4.
Treatment of Fibromyalgia

1. Diagnosis and Reassurance
2. Avoid aggravating factors (cold, narcotics, steroids)
3. Workplace Modification
4. Pharmacological (sleep regulator, NSAIDS)
5. Physical Modalities (heat, massage, sprays, anesthetics, injections)
6. Recreational Aerobic Exercise
7. Patients should take an active role in treatment

Low-impact aerobic exercise should not lead to excessive fatigue or myalgias on the following day, as supramaximal exercise including heavy weightlifting can be associated with increased symptoms and short-term disability.[10] Many fibromyalgia sufferers report a benefit from an aquatic exercise program conducted in a warm pool.

PROGNOSIS

The newly-diagnosed patient with fibromyalgia must be reassured that she/he does not have a life-threatening or disfiguring illness. Most individuals are relieved to learn that their constellation of symptoms is a recognized entity and can be treated effectively.

Recent studies offer hope by demonstrating that many patients with fibromyalgia will have fewer symptoms after sleep has been corrected. Regular physical exercise is also beneficial; in a study of 44 patients enrolled in an exercise program, 47% no longer fulfilled the ACR criteria after two years, and 24% were in complete remission. Despite improvements in sleep and myalgias, some patients will remain chronically symptomatic. Reassurance, counseling, medication and exercise are essential to the long-term management of fibromyalgia.

SUMMARY

Fibromyalgia is characterized by chronic diffuse muscular aches, fatigue and poor sleep; it affects nearly three million individuals in the United States alone, predominantly younger women. The diagnosis of fibromyalgia requires adherence to the American College of Rheumatology criteria and the exclusion of secondary causes and systemic diseases. Treatment with sleep cycle regulators, NSAIDs, and light aerobic exercise is usually helpful. Patients must be reminded that fibromyalgia is often a chronic condition, but can be successfully treated.

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Treatment of Juvenile Rheumatoid Arthritis

Bradley Bloom, MD

JRA: A Separate Entity from "Adult" Rheumatoid Arthritis

Each of us hears in medical school that "children are not just little adults." A corollary is that juvenile rheumatoid arthritis (JRA) is not just rheumatoid arthritis (RA) in juveniles. JRA is in fact at least 3 separate diseases, conventionally denoted as the "subtypes" pauciarticular, polyarticular, and systemic. This trifurcation leads to a diversity in therapeutic approaches. Furthermore, laboratory evidence has shown distinctive differences in the immune and genetic profile of children with JRA versus adult RA. For example, children with pauciarticular JRA commonly have the HLA Class II type DR5 rather than the DR4, and children with systemic JRA have higher levels of many pro-inflammatory than any other condition, including RA.[1]

Historically, academic laboratories and pharmaceutical companies have largely seen JRA as a stepchild of RA. That is, a particular molecular marker may be studied in JRA because it has been found to be abnormal in RA, rather than because it fits in well conceptually with what is known about JRA. Clinical trials have been particularly diffficult in JRA. Again, drugs are not usually used for JRA until/unless they have been proven effective in adults. Furthermore, the drugs are then often only tried anecdotally for quite some time before organized trials are initiated. Even those organized studies have suffered from difficulties in obtaining funding and recruiting subjects.[2] Thus, specific, evidence-based therapeutic interventions in RJA are relatively rare; the "art" of medicine is often in full effect.

How then do physicians devise a treatment strategy for JRA patients? And what progress has been made despite the above limitations?

Traditionally, JRA treatment had always followed the "pyramid" strategy. Recently, however, pediatric rheumatologists have inverted the pyramid along with our internist colleagues, using more aggressive agents earlier in the disease course to prevent destruction and disability. Yet JRA (especially the pauciarticular subtype) is often not of the same malignant ilk as RA, and thus therapeutic strategies need to be tailored to the individual patient. As one rheumatologist put it, "leave pyramids to the Egyptians!"[3] However, the pyramid still provides a useful structure to illustrate new developments in JRA therapy.

> *Methotrexate has clearly become the gold standard for use in moderate to severe disease.*

NSAIDs

Only four of these agents have been approved for use in children: naproxen, ibuprofen, tolmetin, and indomethacin. While these agents are effective, pediatric rheumatologists have not been able to use newer preparations which may be longer-acting, and have less gastrointestinal toxicity. Off-label use of other NSAIDs, however, is common, especially for those with convenient dosing. Likewise, no trials of the new selective COX-2 inhibitors are underway in children, and no pediatric formulations are being devised. Thus it remains likely that for children with milder arthritis, no new options will be available in the near future.

Disease-Modifying Agents

In the past, use of these agents in childhood arthritis was limited for several reasons. First, several of the "milder" agents which have been shown to have low toxicity with modest or moderate efficacy in adults have been shown to be no better than placebo in children; namely, hydroxychloroquine, d-penicillamine, and auranolin.[4] Thus they are not generally used in pediatric arthritis. Intramuscular gold is still sometimes used, and anecdotally effective, but has largely been dropped since the development of methotrexate for uses in JRA, mostly because it involves painful injections.

Abbreviations Used:

JRA	juvenile rheumatoid arthritis
NSAID	non-steroid anti-inflammatory drug
RA	rheumatoid arthritis

Methotrexate has been shown to be highly effective in JRA. As in adults, it is more effective when used in early disease, yet may have dramatic effect even in very severe patients. There is even suggestion in children that use of this drug may lead to cartilage repair. Its once-weekly dosing enhances compliance in children as well. Unfortunately, methotrexate has suffered from an image problem. Initial reports of hepatotoxicity have been proven to be exaggerated. Long-term follow-up studies of children on methotrexate have shown virtually no hepatotoxic-

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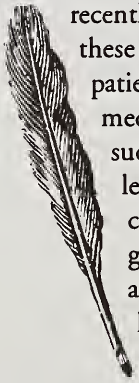
ity by liver biopsy. Pulmonary toxicity, which is rare in adults, is nearly unreported in children. Development of malignancies while taking methotrexate is not increased. Parents and physicians also have been concerned that the FDA only recently approved this medication for use in JRA, yet they don't realize that the approval was held up by a technicality - the fact that the manufacturer had not tested a specific pediatric formulation. Thus in the last few years methotrexate has come into its own as the gold standard for treatment of moderate or severe JRA, yet it is still sometimes difficult to convince families to use it.

Sulfasalazine[5] has long been recognized as effective in RA. Sporadic use has been occurring in JRA for quite some time, but the promising results of a recent open-label trial in children, as well as the report by O'Dell and others that sulfasalazine is an important element of combination therapies for adult RA, have led to more common use, particularly in children whose families resist methotrexate therapy. However, its use in children is hampered by 3-times-a-day use, lack of a readily available liquid preparation, and relatively common occurrences of rashes and gastrointestinal upset.

Leflunomide has not yet been specifically tested in children. However, the suggestion of low toxicity in adults, and benefit to patients who have failed methotrexate therapy, suggests a future role for children as well, especially since no injections are required (a major disadvantage of the new biologic agents discussed below).

IMMUNOSUPRESSIVE AGENTS

Long-term study of patients in Europe who have been treated with azathioprine or chlorambucil have recently documented efficacy of these agents for JRA. However, patients often discontinued the medicine due to side-effects such as nausea; and there is legitimate reason for concern of long-term oncogenic potential of these agents. Thus they will likely be relegated to occasional use only.



Cyclosporine and cyclophosphamide seem to be somewhat more promising therapies, especially for patients with recalcitrant severe systemic disease. Cyclosporine seems particularly effective in reversing the rare but potentially fatal side effect of system disease known as the macrophage activation syndrome, which includes disseminated intrafascular coagulopathy and central nervous sytem changes.[5] Cyclophosphamide has shown modest but definite effects on arthritis activity in children who were otherwise severely disabled, and, more impressively, can reverse long-standing growth failure.[7] However, these agents also carry potential toxicity; and trials have been completely anecdotal.

The most dramatic form of immune suppression is immune ablation, which is achieved through an innuno-ablative cocktail of cyclophosphamide, radiation, etc., followed by autologous stem-cell rescue. Though experimental, this treatment has now been tried in several patients with very severe systemic disease, and in the short-term has led to total remission. However, such treatment carries serious risk of secondary infection, and is extremely expensive.

BIOLOGIC AGENTS

Etanercept (a synthetic soluble TNF receptor analog) is the first biologic agent to be released for use for rheumatoid arthritis. It is also the first second line agent to be tested almost simultaneously in children as in adults, and is thus likely to be apprved in children soon. The pediatric trial showed dramatic response in 70% of trial participants, who were largely children with severe arthritis that had failed treatment with other disease-modifying agents, includng methotrexate.[8] There also seem to be few if any short-term side effects of this medication. Nonetheless, we know that TNF is a necessity for battling infections, and for tumor surveillance, and thus there is concern than long-term use may lead to serious infections or an increased rate of late malignancies.

Several other biologic agents are under development, and it is likely that patients will take "cocktails" of several of these agents in combination. Yet they all have the same drawbacks: extreme expense, delivery by injection (naturally unpopular with children), and the unpredictability of late complications.

NEWER USES OF CORTICOSTEROIDS

Steroids are obviously not a "new" therapy for JRA. However, two uses of these preparations have gathered increasing support in recent years for JRA. The first is intra-articular use. For some time intra-articular injections were barely used in pediatrics, due to concern over damage to developing cartilage. However, increasing evidence has shown that they can be very effective for joints that are resistant to other therapies or for use in patients who have had intolerable side effects with other therapies. Furthermore, their onset of action is extremely rapid, duration of effect in JRA averages 7-11 months (depending on the study), and systemic side effects are minimal.[9] Unfortunately, smaller children often need to be sedated, or as we prefer in our institution, placed under general anesthesia. This therapy is now commonly used in pauciarticular disease, and often in polyarticular disease when one or a few joints remain a problem in a patient who seems to have otherwise responded to a second-line agent.

"Pulse" intravenous steroids (30 mg/kg/dy for 1-3 successive days) are also commonly used in children with JRA, especially under these circumstances: 1) severe systemic disease, such as pericarditis, profound anemia, or high fevers resistant to antipyretics; or 2) severe dysfunction such as inability to walk, despite other therapies.

SUMMARY

Advances in pediatric rheumatology have closely paralleled those in adult rheumatology, but several unique features of JRA have led to some prominent differences. Methotrexate has clearly become the gold standard for use in moderate to severe disease. A variety of the second-line agents and newer methods of corticosteroid dosing can be used in milder disease or in patients who fail or refuse methotrexate. Biologic agents and newer immunosuppressive agents hold out significant promise for those with the most severe disease. As for adult disease, a cure for arthritis is far away, but the lives of these patients have truly been revolutionized by the wide array of newer therapies.

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Advances in Pharmacology

Cox-2 Inhibitors: A New Class of Medication?

Thomas A. Habershaw, Jr., RPH

Non-steroidal anti-inflammatory agents (NSAIDs) are the primary medication utilized to combat arthritis. Since the development of the first NSAID, aspirin, the pharmaceutical industry has developed NSAIDs such as indomethacin, ibuprofen, naproxen and nabumetone with an eye on reducing the frequency of administration and to reduce the gastrointestinal adverse effects, renal toxicity and bleeding exhibited by the class as a whole. In many cases, any of these adverse effects can be severe enough to stop therapy.

While the exact mechanism of action of the NSAIDs is unclear, it appears that their analgesic and anti-inflammatory actions are due chiefly to the inhibition of prostaglandin synthesis. This is accomplished through the inhibition of cyclooxygenase (COX) enzymes necessary for prostaglandin synthesis. There are two COX isoforms which have been identified: COX-1, which is found in most tissues and is believed to protect the gastric mucosa. COX-2 is found chiefly in the brain and kidney, but also in other tissues and at inflammation sites. COX-1 is found in large amounts in the platelets while COX-2 is not. Both are found in the synovial fluid of patients with rheumatoid arthritis.

> *Since COX-2 inhibitors do not affect cyclooxygenase-1, the occurence of severe gastrointestinal upset and complication is less than those of the other NSAIDs.*

Older NSAIDs block both of the COX enzymes. While classified by the Food and Drug Administration (FDA) as another NSAID, a new type of medication has been marketed which specifically targets the COX-2 enzyme. The COX-2 inhibitors exhibit the anti-inflammatory, analgesic and antipyretic effects seen with the other NSAIDs. However, as the name indicates, cyclooxygenase-2 is specifically targeted and cyclooxygenase-1 is left alone. Since COX-1 serves to protect the stomach, the gastrointestinal side effects are minimized. The first COX=2 inhibitor, marketed in early 1999, is Celebrex® (celecoxib), followed in late spring 1999 by Vioxx® (rofecoxib). This article will focus on celecoxib.

Pharmacokinetics

Peak plasma levels of celecoxib occur within 3 hours of administration, with a steady state reached by day 5 of therapy. Food has little effect on absorption so administration can be scheduled without regard to meals. Metabolism is performed through the cytochrome P450 2C9; patients who are known or suspected poor P450 2C9 metabolizers should be administered COX-2 inhibitors with caution. With a half-life of approximately 11 hours, 37% of the drug is excreted in the feces and 27% in the urine.

Abbreviations Used:

COX	cyclooxygenase
FDA	Food and Drug Administration
NSAID	non-steroidal anti-inflammatory drug

Dosing[1]

In the treatment of osteoarthritis, the recommended dosage of celecoxib is either 100 mg P.O.B.I.D. or 200 mg P.O. daily. In the treatment of rheumatoid arthritis, the recommended dose is 100-200 mg P.O.B.I.D.

Patients with moderate hepatic insufficiency should receive low doses; patients with severe hepatic impairment should not be treated with celecoxib. Patients with mild to moderate renal insufficiency do not require lower doses; however, patients with severe renal failure should not be treated with celecoxib.

Clinical Indications

While the COX-2 inhibitors have been classified by the FDA as NSAIDs, they have not been given all the approved indications of the older NSAIDs. At present, celecoxib is indicated only for the treatment of osteo- and rheumatoid arthritis. [2,3] Rofecoxib is indicated for the treatment of osteo-arthritis and dental

MEDICATION	DOSE/DAY	COST/DAY*
Celecoxib	100 mg BID	$2.36
Ibuprofen	600 mg QID	$0.20
Naproxen	500 mg BID	$0.40
Nabumetone	500 mg BID	$1.86
Diclofenac	50 mg BID	$0.90
Indomethacin	25-50 mg BID	$0.24
Sulinac	150 mg BID	$0.30
Rofecoxib	25 mg QD	$2.04

Table
Adverse Events Occuring in ≥ 2% of Celecoxib Patients

	Celebrex (100-200 mg BID) and (200 mg QD) (N=4146)	Placebo (N=1864)	Naproxen 500 mg BID (N=1366)	Ibuprofen 800 mg TID (N=387)	Diclofenac 75 mg BID (N=345)
Gastrointestinal					
Abdominal pain	4.1%	2.8%	7.7%	9.0%	9.0%
Diarrhea	5.6%	3.8%	5.3%	9.3%	5.8%
Dyspepsia	8.8%	6.2%	12.2%	10.9%	12.8%
Flatulence	2.2%	1.0%	3.6%	4.1%	3.5%
Nausea	3.5%	4.2%	6.0%	3.4%	6.7%
Body as a whole					
Back Pain	2.8%	3.6%	2.2%	2.6%	0.9%
Peripheral edema	2.1%	1.1%	2.1%	1.0%	3.5%
Injury-accidental	2.9%	2.3%	3.0%	2.6%	3.2%
Central and peripheral nervous system					
Dizziness	2.0%	1.7%	2.6%	1.3%	2.3%
Headache	15.8%	20.2%	14.5%	15.5%	15.4%
Psychiatric					
Insomnia	2.3%	2.3%	2.9%	1.3%	1.4%
Respiratory					
Pharyngitis	2.3%	1.1%	1.7%	1.6%	2.6%
Rhinitis	2.0%	1.3%	2.4%	2.3%	0.6%
Sinusitis	5.0%	4.3%	4.0%	5.4%	5.8%
Upper resp. tract infection	8.1%	6.7%	9.9%	9.8%	9.9%
Skin					
Rash	2.2%	2.1%	2.1%	1.3%	1.2%

In placebo- or active-controlled clinical trials, the discontinuation rate due to adverse events was 7.1% for patients receiving CELECOXIB and 6.1% for patients receiving pacebo. Among the most common reasons for discontinuation due to adverse events in the CELECOXIB treatment groups were dyspepsia and abdominal pain (cited as reasons for discontinuation in 0.8% and 0.7% of CELECOXIB patients, respectively). Among patients receiving placebo, 0.6% withdrew due to abdominal pain.

pain. Neither is currently approved for generalized analgesia, muscle pain and headaches. Further clinical data have been required by the FDA prior to approval for additional indications. These studies will also yield further data as to how the COX-2 inhibitors compare therapeutically to the older NSAIDs.

DRUG INTERACTIONS

Since celecoxib is metabolized through the P450 2C9 pathway in the liver, medications which inhibit 2C9 should be used with caution in combination with a COX-2 inhibitor. During clinical studies, concomitant administration of celecoxib and fluconazole led to the doubling of celecoxib plasma levels; when used in patients receiving lithium therapy, celecoxib increased levels of lithium. Interactions occur between NSAIDs and ACE inhibitors, furosemide and thiazides, leading to a decrease in the antihypertensive actions of these medications. Use with aspirin may lead to an increase in gastrointestinal ulcers and other problems.

ADVERSE REACTIONS

COX-2 inhibitors have produced fluid retention and edema in trials and should be used with caution in patients with asthma and should not be used in patients with aspirin-induced asthma. Since COX-2 inhibitors do not affect cyclooxygenase-1, the occurence of severe gastrointestinal upset and complication is less than those of the other NSAIDs.

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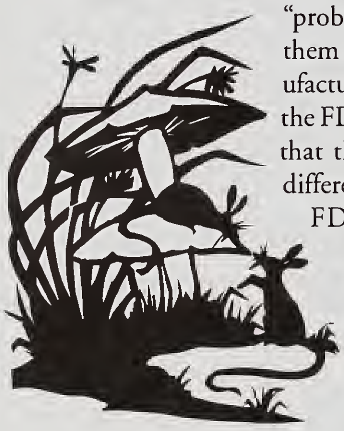
During clinical trials, approximately 7% of the patients dropped from the study, primarily due to abdominal pain and dypepsia.[3] Table 1 highlights the comparison of adverse reactions of celecoxib and some of the more common NSAIDs.

CONCLUSIONS

Since the release of the first COX-2 inhibitor (celecoxib) in January 1999, physicians have written hundreds of thousands of prescriptions. The drawback to NSAID therapy for many patients has been the adverse reactions produced by the older NSAIDs; in many cases the severity of these adverse reactions resulted in cessation of therapy, leaving few alternatives to patients and their physicians. The promotional feature of the COX-2 inhibitors is the decrease in the occurence and severity of these reactions.

However, the COX-2 inhibitors are not without their "problems." The FDA classifies them as NSAIDs. To date, manufacturers have failed to convince the FDA with their study evidence that the COX-2 inhibitors are a different class of medication; the FDA has required evidence from long-term studies. [4] Consequently, many health care insurers have either not paid for or restricted the coverage of COX-2 inhibitors.[5] Further, a recent study at the University of Pennsylvania Medical Center looked at the pharmacology of the COX-2 inhibitors and found aspects that could increase the risk of heart attacks, strokes, and other cardiovascular problems.



The COX-2 inhibitors are expected to be a boon to arthritis therapy. As with any other new medication, long-term use will provide evidence as to their risk-to-benefit ratio.

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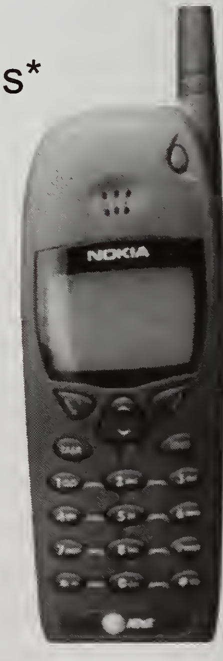

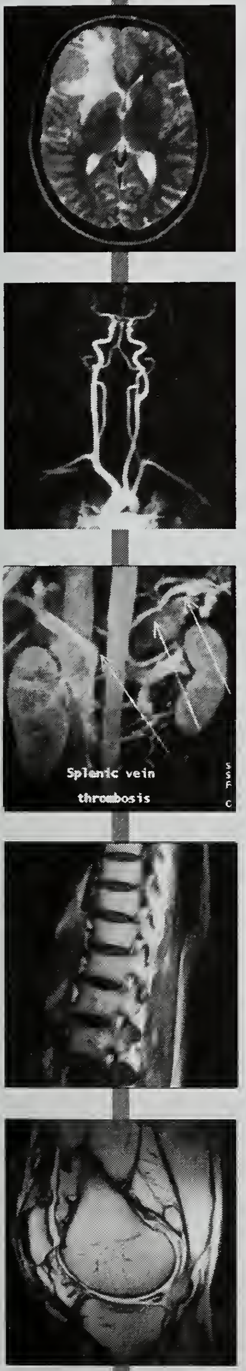

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Outpatient Disease Management: RIQP Wants You!

Raymond Maxim, MD

Rhode Island Quality Partners (RIQP) needs you! ... to help. Over the past two and a half years, great effort either in terms of people and/or money has been put forth by the hospitals, Rhode Island Medical Society on its own and in partnership with RIQP, HCFA and others to improve the quality of health care given to Medicare beneficiaries. The vast majority of the past efforts have been focused on inpatient care where health care expenditures are high, medical records are accessible, and the processes of care are relatively easy to evaluate. Efforts to evaluate care processes have led to care maps or clinical pathways and changes in systems to improve the efficiency and effectiveness of the delivery system and they have been fairly successful in most cases. With these successes has come the realization that in many chronic diseases the single most important predictor of long term outcome following admission to the hospital is the subsequent outpatient care. Keeping this in mind it seems prudent to extend from the hospital into the outpatient arena.

While inpatient care may be the most expensive portion of our patients' care, it may be the least important as measured by the long-term relationships we develop with our patients. It is no secret that most of medicine happens outside the walls of the hospital and in the confines of our offices. As a consequence, the quality of care our patients receive out of the hospital significantly affects what happens to our patients in the hospital or if they even get admitted at all! Unfortunately, the forces driving medicine have led us away from prevention to episodic care. We see patients, for the most part, when they have a problem and have very little time to counsel about healthy lifestyles, manage their chronic disease or screen for early disease.

On the inpatient side, almost every network, hospital, or health plan has its own version of each pathway. A similar scenario is happening in outpatient care. We all receive guidelines for managing diabetes care, cardiovascular disease, and other common conditions from health plans. Each of these guidelines is slightly different from the other and from nationally recognized guidelines. This inevitably leads to the inability to follow any guidelines consistently, and the printed guidelines end up in the "circular" file where they can be of little help to anyone.

Abbreviations Used:

CHF	congestive heart failure
DEARS	Diabetes Education Assessment Referral and Screening
HCFA	Health Care Financing Administration
MCO	managed care organization
RIQP	Rhode Island Quality Partners

What RIQP is attempting to do is to work with the local health plans to develop a universal set of evidence-based guidelines. To their credit, the MCOs have been receptive to the suggestion and have made their willingness to work with us very clear. If this is accomplished, it will be easier to provide higher levels of preventive care and screening for our patients. However, this also means that we need to step up our efforts as a profession and as individual practitioners to participate in the decision-making process to define these guidelines and then to improve care accordingly.

So what is it that RIQP wants from you? We would like your help in improving the delivery of outpatient care. It is as simple and as complicated as that.

HCFA has charged peer review organizations with improving the care in a number of high morbidity and mortality conditions. These include several outpatient topics, a few that cut across the inpatient and outpatient settings, and some that are purely hospital topics. They will seem familiar to you as they are projects that RIQP has done in the past.

Congestive heart failure and anticoagulation in atrial fibrillation to prevent stroke are two of the topics that bridge the gap between the inpatient and outpatient environment. The literature is clear that anticoagulation with warfarin in patients with atrial fibrillation reduces the incidence of stroke. This means that the patient is not hospitalized, there is comparatively little expense in treating these patients and our patients do not have to suffer the indignities associated with having a stroke. Readmission following hospitalization for CHF can be reduced with good attention to ACE inhibitors, their use and dose; sodium and weight management. In Rhode Island the readmission rate for CHF within thirty days is between 25% and 30%. The difficulty with CHF is the transition from hospitalization where the patient receives 24-hour care to home, where the patient may have little or none of this support. This will be a focus area where we can make the greatest advances.

The outpatient conditions we are addressing are influenza and pneumococcal immunizations, breast cancer screening, and diabetes care. In the past, we have provided individualized rates for pneumococcal vaccination to primary care physicians. This led physicians to examine their own practices and make changes. We intend to provide the same service for influenza and mammography screening. As always, the information provided is for personal benchmarking and will not be shared with anyone other than the individual practitioner. Our only request is that you take this information and put it to good use. If your rates of influenza vaccination are low, examine your practice or system to provide immunization. It is likely that a simple change in how things are done in your office may improve your rates dramatically. A number of physicians I have talked with were not billing properly and were not getting credit for services they provided. They were also not getting paid for services they had provided!

Diabetes is a disease that will be addressed more aggressively in the next three years. In the past we have limited ourselves to measuring the rates of funduscopic exams obtained by beneficiaries with diabetes. Now we will be looking at all aspects of diabetes care. The goal is to prevent long term complications associated with diabetes. To that end, we are working with the Rhode Island Department of Health and their Diabetes Control Program, called DEARS (Diabetes Education Assessment Referral and Screening). This is a comprehensive program that I have piloted in my own office with great success. How it works is a provider requests the DEARS team to come to his or her office on a specific day. Invitations are sent to the diabetic patients to attend a complimentary breakfast and education session. On the scheduled morning a team of Certified Diabetes Outpatient Educators that includes a nurse, dietitian, and pharmacist, will arrive at the office along with a phlebotomist and possibly an ophthalmologist. They will provide the necessary counseling, draw the appropriate laboratory work and provide a dilated eye exam if needed. The clinician has the opportunity to address the patients as a group as well as provide individual exams and counseling based on the lab results and other information collected from the patients that day. The opportunity is there to bill for the services provided by the clinician.

Finally, for most physicians we will provide data that reflect their performance compared to the norm so that they can benchmark themselves against others. For other clinicians we will provide individualized training in their offices in an effort to help them improve their performance (for CME credits, of course). During these visits the clinician will not only have an opportunity to discuss aspects of care with an expert clinician or clinicians, they will have the opportunity to offer suggestions about how we can help physicians provide even higher quality medical care. These visits for RIQP will be both information-disseminating and fact-finding. We look forward to working closely with you over the next three years. We need you and hope that you will answer our call.

As always please contact us with questions or comments.

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Health by Numbers

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Arthritis and Disability in Rhode Island

Hanna Kim, PhD, and John P. Fulton, PhD

Almost one-sixth of the population of the United States suffers from one of the many diseases and conditions subsumed under "arthritis," a great catch-all of medical terminology. In the U.S. as a whole, arthritis leads all other chronic diseases and conditions as a cause of disability (defined by the World Health Organiztion as an "impairment" at the level of bodily functioning, an "activity limitation" at the level of personal functioning, or a "participation restriction" at the level of social functioning).[1]

Although arthritis affects people throughout the life span, its prevalence increases greatly in later life. Among persons ages 65 and over, one-half are beset with arthritis in one of its many forms.[1] Thus arthritis constitutes a greater share of the disease burden as a population ages. In the early 21st century this burden will reach an unprecedented magnitude in developed countries, as the large cohort of persons born after the close of World War II becomes elderly.

To prepare for the increased importance of arthritis in the United States, the Arthritis Foundation, the Association of State and Territorial Health Officials, and the Centers for Disease Control and Prevention (CDC) collaborated on *The National Arthritis Action Plan,* published in January, 1999. The *Plan* has been distributed widely in a bid to stimulate planning at the state and local level, where much of the work to control this epidemic must occur.

In parallel with this endeavor, the Rhode Island Department of Health (HEALTH) has monitored the burden of arthritis in our state, including the relationship between arthritis and disability, to prepare the State for planning and intervention in the years ahead.

Methods

In 1996 and 1997 HEALTH incorporated an arthritis surveillance module in the Rhode Island Behavioral Risk Factor Surveillance System (RI BRFSS) survey, an ongoing telephone survey of adults which measures health risks, health-related behaviors, health problems, and health care utilization. The data presented here will focus on the 1,842 responses obtained in the 1997 survey.

Abbreviations Used:

CDC	Centers for Disease Control and Prevention
HEALTH	Rhode Island Department of Health
R	respondent
RI BRFSS	Rhode Island Behavioral Risk Factor Surveillance System

Respondents who answered "Yes" to the question, "Have you ever been told by a doctor that you have arthritis?" were classified as people who "have arthritis." This approach yields a conservative estimate of the number and proportion of respondents who actually suffer from arthritis, but obtains more valid responses than those based on respondents' personal definitions of arthritis.

Four indicators were used to paint disability with a broad brush: 1/ "Poor Health" (Health is rated by respondent ("R") as "fair" or "poor"); 2/ "Limited Activity" (R's usual activity - job, school, or housework - is limited); 3/ "Chronic Pain" (Pain made it hard to perform R's usual activity for eight or more days in the past month); 4/ "Dysphoric Mood" (R felt sad or blue for eight or more days in the past month). The percentages of respondents who met each of these criteria

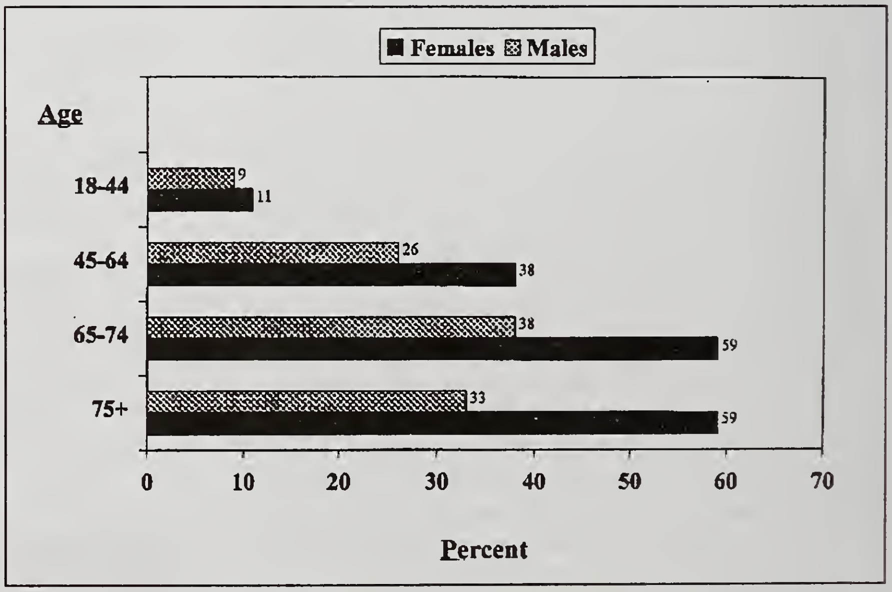

Figure 1. Prevalence of arthritis by age and sex, Rhode Island, 1997.

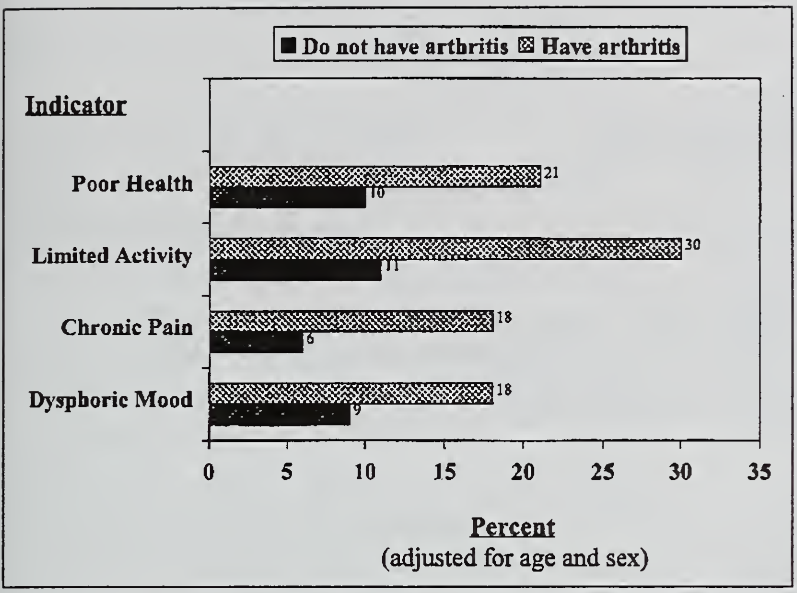

Figure 2. Disability by arthritis status, Rhode Island, 1997.

were adjusted for age and sex, to remove the effects of these variables on comparisons by arthritis status. Collectively, all respondents to the 1997 RI BRFSS were used as the "standard population" in the adjustment. Four broad age-groups were employed: 18-44, 45-64, 65-74, and 75+.

Data on obesity, a modifiable risk factor for arthritis, were worked up similarly, adjusting for age and sex. Obesity was defined as having a body mass index of 30 or greater.

Results

Overall, 24% of respondents (ages 18 and over) have arthritis. Extrapolating to people of all ages, the percentage of all Rhode Islanders who have arthritis is estimated to exceed 18% (more than 181,000 of the 984,000 people estimated to be living in Rhode Island in 1997).

About the same proportion of non-Hispanic whites and non-Hispanic blacks have arthritis. A smaller proportion of Hispanics have arthritis, which reflects the younger age structure of this population in Rhode Island. (The 1997 RI BRFSS did not survey sufficient numbers of Asians and Native Americans to estimate the prevalence of arthritis in these groups accurately.)

In Rhode Island, as elsewhere in the U.S., the prevalence of arthritis increases with age, from about 10% among people ages 18-44 to about 35% among men ages 65 and over and about 59% among women ages 65 and over (Figure 1). Arthritis is more common among women than men over the life span.

About half the respondents who have arthritis reported the type of arthritis a health care provider said they had: osteoarthritis - 28%; rheumatoid arthritis - 12%; rheumatism - 6%; Lyme disease - <1%; other specific types - 7%; don't know - 46%.

Even after age/sex adjustments, people with arthritis were much more likely than others to report poor health, limited activity, chronic pain, and dysphoric mood (Figure 2), and to meet the criterion for obesity: 20% of those with arthritis were obese at the time of the survey, compared to 12% of other respondents.

Discussion

Arthritis affects more than 18% of Rhode Island's 984,000 residents. As the "baby boom" generation ages, the proportion of people with arthritis will increase.

Arthritis causes significant physical and emotional disability. People with arthritis were much more likely than others to report a problem with health, the performance of usual activities, pain, and mood. Thus, as the proportion of people with arthritis grows in the 21st century, so will the proportions of people with physical and emotional disabilities.

Fortunately, the risk of arthritis may be reduced in four ways: by reducing obesity, by avoiding joint injuries, by controlling Lyme disease and other infections, and by redesigning work activities to reduce chronic, repetitive stresses to the joints.

Obesity is a good place to start. Although intractable in some, it can be avoided with sensible eating and moderate daily exercise. Both of these behaviors are within our grasp as a society. Thus far, Rhode Island has achieved moderate success in mobilizing itself on both fronts, through HEALTH's "Five-A-Day for Better Health" program and the Prevention Coalition's "Move It!" campaign. HEALTH has also recently submitted a grant proposal to the CDC for a program to help focus the community on these issues.

Physicians play an essential role in the promotion of healthy lifestyles, guiding their patients toward sensible eating and exercise, and supporting them medically and emotionally in the process. But are the physicians themselves supported in this work? Are they afforded sufficient time to counsel patients? These are serious concerns in 1999. Imagine how the importance of these concerns will increase as the population ages, increasing the burden of arthritis and its associated disability, and intensifying the need for health promotion in physicians' offices. As a society, we must come to grips with this challenge, and the sooner, the better.

Dr. Hanna Kim, PhD, is Public Health Epidemiologist, Division of Family Health, Rhode Island Department of Health.

John P. Fulton, PhD, is Acting Associate Director, Division of Disease Prevention and Control, Rhode Island Department of Health, and Clinical Associate Professor of Community Health, Brown University School of Medicine.

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Public Health Briefing

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

Reducing the Burden of Arthritis

David Hamel, MPA, and Andrea G. Vastis, MPH, CHES

The Problem

Data from the 1997 RI Behavioral Risk Factor Surveillance System (RI BRFSS) reveal two sobering facts about the prevalence of arthritis in the Rhode Island population:

- About 30% of the respondents (ages 18 and over) had experienced pain, aching, stiffness, or swelling of the joints in the previous 12 months, and
- About 25% had been told they have arthritis by a physician.

In addition, respondents who had been told they have arthritis were far more likely than other respondents to report that they are limited in performing their major activity (job, school, or housework), and were more likely to be obese.[1] Given the prevalence and severity of arthritis in Rhode Island, an arthritis program to reduce the burden of these diseases and conditions on the State's population is a necessary part of our public health efforts.

A National Plan

The National Arthritis Action Plan, A Public Health Strategy, was jointly developed by the National Arthritis Foundation, the Association of State and Territorial Health Officers, and the Centers for Disease Control and Prevention (CDC).[2] The plan describes a comprehensive approach to reducing the burden of arthritis in the U.S., and provides guidance for states developing their own action plans, recommending attention to:

- Surveillance, epidemiology, and prevention research,
- Communication and education,
- Programs, policies and systems, and
- Public health partnership with health care providers.

Rhode Island's Approach

In June 1999, the Rhode Island Department of Health (HEALTH) engaged a few key community partners in preliminary planning to reduce the burden of arthritis in Rhode Island, using the National Arthritis Action Plan[2] as a basis. This brief activity led to the development of a proposal to the CDC for a state Arthritis Prevention and Management program. The proposed four-year program would:

- Assemble a core advisory committee,
- Convene essential community partners to develop a state arthritis action plan,
- Enhance arthritis surveillance,
- Develop a communications strategy for the public and health care providers, and
- Undertake patient and provider education projects, with two main goals:
    - To reduce the prevalence of arthritis symptoms in the Rhode Island population, and
    - To improve the health and functional status of people who have arthritis in its various forms.

Abbreviations Used:

BRFSS	Behavioral Risk Factor Surveillance System
CDC	Centers for Disease Control and Prevention

In conformity with the National Arthritis Action Plan, the proposed program incorporates the following strategies to reduce the burden of arthritis in the State:

- Promoting weight control,
- Preventing injuries,
- Preventing Lyme disease,
- Diagnosing arthritis early,
- Treating arthritis according to best practices, and
- Promoting self-management for people with arthritis.

The Role of Physicians

Physicians will play a key role in developing Rhode Island's state arthritis action plan, defining the role of the medical profession in the prevention and management of arthritis, and guiding other community partners in their collaboration with physicians. In the interim, physicians may wish to note a few essential recommendations from the National Arthritis Action Plan for medical care providers:

- Assure patients that arthritis is not a normal part of aging, nor is it exclusive to elders,
- Promote self-management and non-pharmacological interventions with patients,
- Promote weight loss, if appropriate, through sensible eating and moderate physical activity,
- Refer patients to self-management programs run by the Arthritis Foundation and other agencies for additional education and support, and
- Follow best practices such as may be found in Clinical Care in the Rheumatic Diseases (from the Association of Rheumatology Health Professionals).

The caring practitioner's personal attitude toward arthritis and aging is arguably the biggest factor for effective treatment of patients with arthritis. Working with patients as partners in arthritis management will enhance their self esteem and help them develop a sense of control over the course of their illness.

Providers may wish to refer patients to the Arthritis Foundation for information on arthritis, self-management, and support groups: The Arthritis Foundation, 820 Park Avenue, Cranston, RI 02910; Tel: (401) 461-9339; Fax: (401) 461-0774

COMMENTS?

We invite your input on the proposed public health approach to arthritis prevention and control for Rhode Island. Please send comments and questions to: Andrea Vastis, 3 Capitol Hill (Room 409), Providence, RI 02908-5097; fax: 401-222-4415; e-mail: :AndreaV@doh.state.ri.us

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1. Kim H, Fulton JP. Arthritis and disability in Rhode Island. Health by Numbers. *Medicine & Health / RI* 1999;82:338-9.
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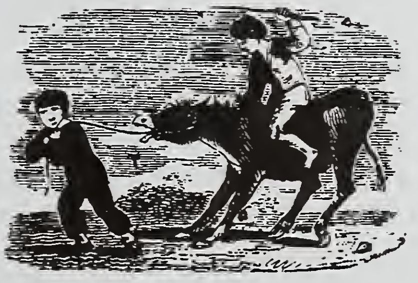

Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	Sept. 1998	12 Months Ending with Sept. 1998		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	222	3,162	319.3	3,823.5
Malignant Neoplasms	204	2,523	254.8	6,971.0**
Cerebrovascular Diseases	35	618	62.4	751.5
Injuries (Accident/Suicide/Homicide)	34	360	36.4	7,065.0
COPD	24	469	47.4	427.5

Vital Events	Reporting Period		
	February 1999	12 Months Ending with February 1999	
	Number	Number	Rates
Live Births	1,285	13,490	13.6*
Deaths	880	9,692	9.8*
Infant Deaths	(8)	(97)	7.2#
Neonatal deaths	(5)	(73)	5.4#
Marriages	311	7,454	7.5*
Divorces	328	3,296	3.3*
Induced Terminations	479	4,717	349.7#
Spontaneous Fetal Deaths	100	1,072	79.5#
Under 20 weeks gestation	(94)	(983)	72.9#
20+ weeks gestation	(6)	(89)	6.6#

* Rates per 1,000 estimated population # Rates per 1,000 live births

**Excludes one death of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 990,225

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

Book Review

The Tapestry of Medicine: Collected Essays from *The Providence Journal-Bulletin* and *Medicine & Health/Rhode Island*

by Stanley M. Aronson, MD

reviewed by Stephen Davis, MD

I liked this book because it was interesting.

So begins many a book report.

This book is *The Tapestry of Medicine* by Dr. Stanley Aronson, Dean Emeritus of the Brown University School of Medicine, former editor of *Medicine & Health/Rhode Island*, and local, as he describes Noah Webster, polymath.

But this book really is interesting. I knew that it was interesting because when I was reading it at bedtime, I did not fall asleep. Its three-to five-page chapters lead to the "just one more before I stop" phenomenon. They're like potato chips.

This book is also thoughtful, somewhat provocative, edifying, stimulating, amusing, and, especially, educational. It is a lot like its author.

This is a book that just had to be compiled; the essays of Dr. Stanley Aronson, AKA the sage of Elm Street, had lingered too long as separate entities; their compilation has intensified their literary effect. While reading these elegant portrayals of the early (and we mean "early"!) days of medicine, of curious and quaint times passed, of medical heroes and heroines, and of other significant mythological, Biblical, and antediluvian events that hardly anyone else knows about but that have shaped our present world, it is not too long before one starts to wonder "How does he know all this stuff?"

A colleague close to me had a theory. She suggested, "He makes it up."

I checked it out.

Wrong.

The stories of pellagra and scurvy are right on; the few details with which I had been familiar prior to reading his fascinating renditions were accurately portrayed.

This collection has 73 essays grouped into nine thematic sections. Typical section titles are "The Growth of Medicine in Western Europe" (9 essays), "The Roots of American Medicne" (15 essays), "The Blessings of Sight" (4 essays), and "The Primitive Art of Prophesy" (7 essays). My favorite essays were those of the history of medicine, and those with etymological tours de force, such as "How They Named the Vitamins".

If you like words, then you will love this book. Tell your children to read it before taking their SATs or MCATs and their verbal section scores will increase. Where else do you find delightful words like "lunambulism", "hepatoscopy", "omphalomancy", and "miasmatists"? Where else do you find provocative phrases such as "the iatrochemical school of medical theory" and "e-mail...gravitates towards the graffiti zone of correspondence"? Not many tomes connect the roots of the words for "alma mater", "matriculate", "matrimony", and "endometrium" as this one does. Where else can you casually learn the origin of words such as "bedlam", and find out what is the epitaph on Shakespeare's tomb? Where else can you find such elegant writing about the gall bladder ("Sublimely exalted power has given way to chopped liver,") and the pancreas ("...and the memory of an obscure, martyred youth somehow merged into an organ of unsurpassed complexity")?

These essays are delightful and informative reading for both physicians and non-physicians. For physicians it will remind us of the complexity and fascination of medicine and also remind us that those same characteristics apply to the non-medical world. This book will make a great present for carefully-chosen recipients.

The format, content, and style of *The Tapestry of Medicine* make for excellent bedtime reading; however, be forewarned: it won't put you to sleep.

And to top it all off, proceeds from the book are generously contributed to the Brown Medical School.

Stephen Davis, MD, is Clinical Associate Professor, Department of Family Medicine, Memorial Hospital of Rhode Island.

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NINETY YEARS AGO

[SEPTEMBER, 1909]

Franklin Clark, MD, discussed "The Problem of Centenarianism." Previous studies had shown that women and married people lived longest. Culling the Registration Reports of Rhode Island from 1852 to 1892, Dr. Clark found corroborating evidence for the longevity of women: "women, married or single, outlive the opposite sex." The 1875 Rhode Island census included 2,102 widowers, and 7,926 widows. As for marriage ("the conjugal condition is the natural state of not only man but of all living beings"), that too contributed to longevity, though Dr. Clark conceded that self-selection may have been an intervening variable. Finally, Dr. Clark judged heredity (he came from "a pure New England ancestry") key: "It is a well-known historical fact that the ... laboring classes are not long-lived."

In an Open Letter to readers, James Krauss, MD, announced the formation of a Scientific Clinical Research Society, to meet at the Boston Medical Library. This new organization sought: 1) to establish an American Association of Clinical Research, 2) to spur research in hospitals, and 3) launch an American Journal of Clinical Research. Dr. Krauss notes, "The crux of the matter is that lab proof is not sufficient clinical proof."

FIFTY YEARS AGO

[SEPTEMBER, 1949]

In "Diagnostic Techniques for Children with Cerebral Palsy" - written to mark the first year of Meeting Street School - Dr. Eric Denhoff urged physicians to communicate honestly with parents. Indeed, the detailed medical histories of Meeting Street patients revealed physician obfuscation, leading to parental anger. "From the histories, one is impressed by the antagonism of parents towards the medical profession, principally because many were lulled into believing that their child would develop normally...." At the same time, Dr. Denhoff stressed the need for early diagnosis, so that "the family can be helped by their doctor to readjust their lives around a crippled child." He summarized data on the 37 children treated at Meeting Street School; e.g., the average age of mother was 28.2 (higher than nationally), 36% of the children were born premature, 67% had some abnormal symptom at birth or shortly afterward.

An editorial summarizing the report of the Children's Health Service (compiled in 1946 and 1947), contrasting it with state pediatric data, cited a major failing: the minimal training in pediatrics. Nationally, 90-95% of pediatric care was given by general practitioners, who spent 31% of their time caring for children; yet pediatric training was "inadequate."

A second editorial criticized physicians' willingness to certify convicted bookmakers as too ill for jail: "The press opines that bookmaking must be one of the hazardous occupations judging by the high percentage of sick men found in it." Physicans should be "concerned that we not get the reputation of beling obliging persons willing to help law-breaking bookies."

TWENTY FIVE YEARS AGO

[SEPTEMBER, 1974]

In "A Message from the Dean," David Greer, MD, Associate Dean of Medical Affairs, described the embryonic family medicine residency program at Memorial Hospital - spurred by "community pressure stimulated by an awareness of the progressively decreasing number of primary care physicians in the nation."

Francis Horn (president of URI, 1958-67), discussed "The Voluntary Health Agency - Is It Still Necessary?" at a dinner meeting of the Rhode Island Lung Association and the Rhode Island Thoracic Society. Dr. Horn responded, "Yes," because of several factors: the uncertainty of government support, the danger of a growing federal bureaucracy, the flexibility of a private-sector organization, and "the opportunity for the average American to exercise his deep-seated commitment to volunteerism."

After analyzing 16 randomly selected cases of sensorineural hearing loss at the Rhode Island Hospital Language and Speech Center, J. Barry Regan, EdD, and Janet Tobin, MS, concluded acupuncture therapy ineffective. ("Acupuncture: Effectiveness as a treatment of sensorineural hearing loss")

In "Emergency Medical Services and the RIMS," Edward Lynch, MA, described steady annual expansion of training for EMS technicians: the first class in 1970 at Miriam had 34 students; the spring 1978 class enrolled 285. Each course was coordinated by a physician member of the Emergency Medical Services Committee of RIMS who was also on staff at the hospital.

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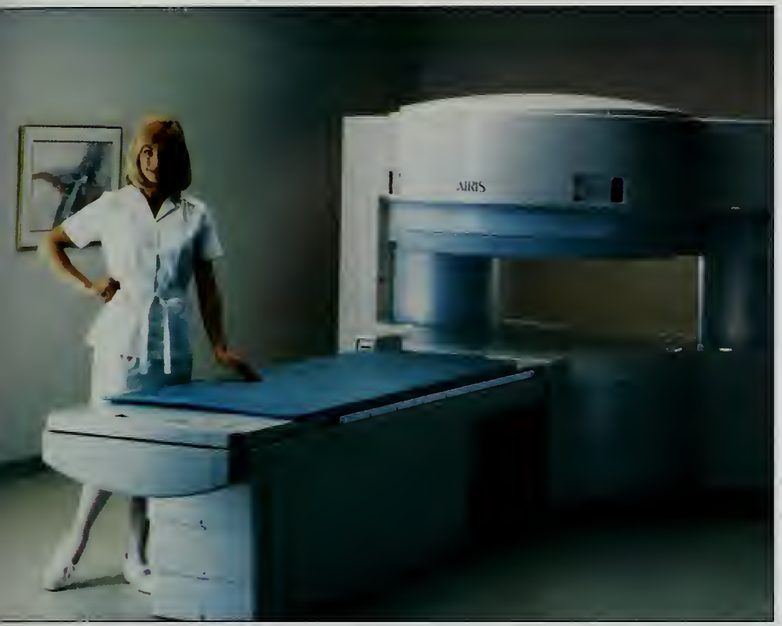

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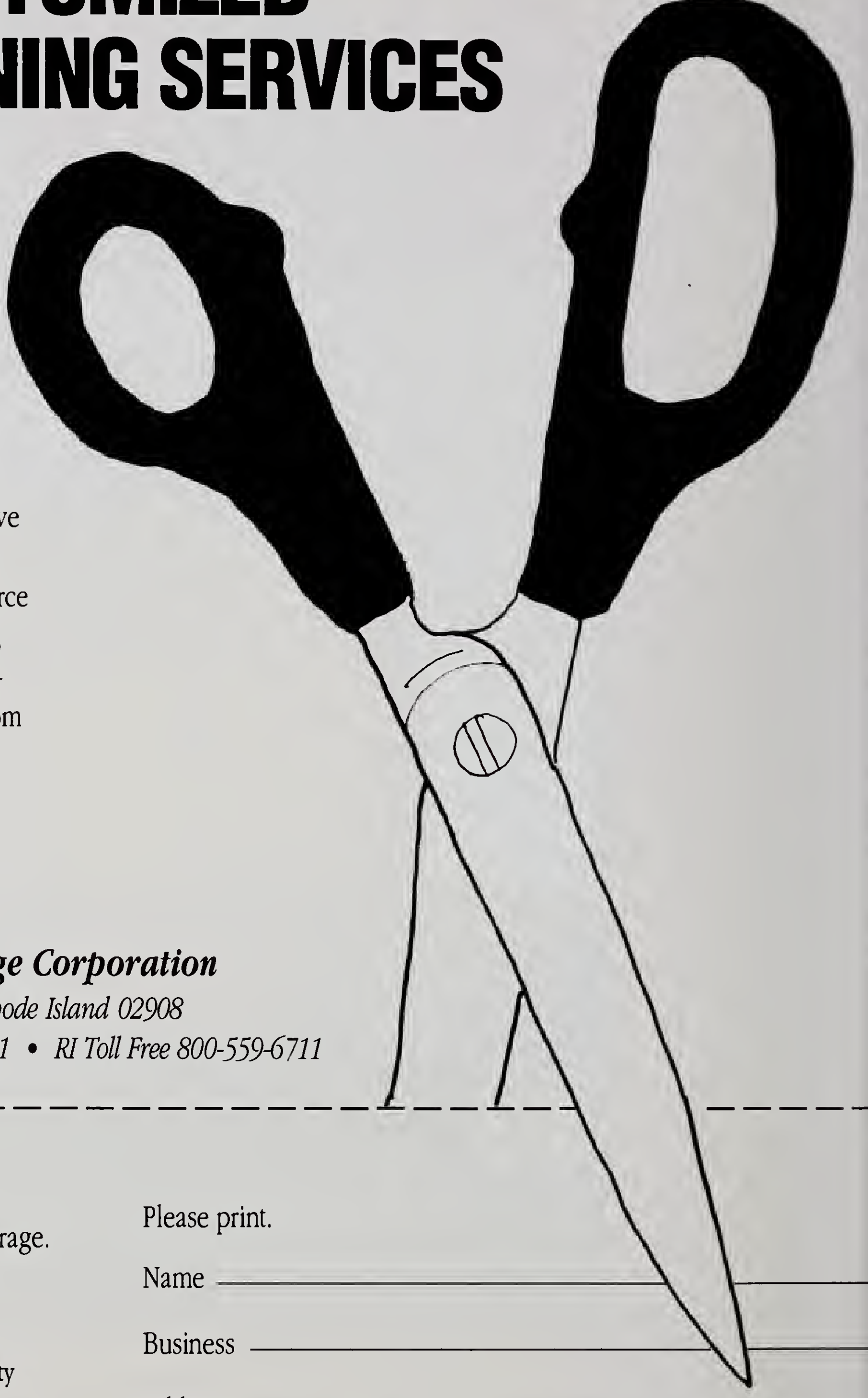

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COMMENTARIES

Vital Signs Stable

When I was a resident physician, we would prepare the medical students who presented cases to the department chair, indicating to them which terminology was acceptable and which not. The departmental chair was notorious for his intolerance of trite and needlessly elegant parlance. Patients were “men” and “women.” To describe a patient as a “male” was to incur the response: “Of what species?” Race was not to be mentioned, unless relevant to the medical condition of the patient, as in the case of a possible stroke in a young person where sickle cell disease might be a precipitating factor. Limbs were described by name. “Upper extremity” was eschewed in favor of “arm,” and “lower extremities” were called “legs”. “Past medical history” was needlessly redundant.

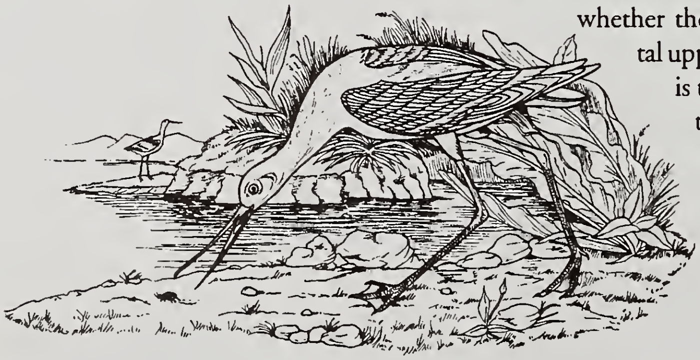
The clinical chief at the other hospitals where these residents worked was equally firm in his requirements, although those requirements were different. Both chiefs were smart and talented, widely recognized for their clinical acumen and teaching skills; both were authors (not just editors) of major texts and superb editors.

What was the point of their scrupulous insistence? They were seeking concise unambiguous communication and the avoidance of distracting, unnecessarily verbose information. They demanded that each case presentation have a clear beginning, middle and end, containing the appropriate “negatives” (did not have fever, malaise, weight loss). The presentation, in their judgment, was an editorial statement, a distillation of a lengthier compilation of historical and diagnostic material in the patient’s chart, which was filled with redundancies and irrelevancies. It was to be offered in the form of a story that led the listener to the same conclusion as the presenter.

When in training, I wondered if I was going to do this too, or be more accommodating and avoid being labeled a curmudgeon. Or might I not care enough? Would certain phrases bother me enough to interrupt? Would I insist on not allowing phrases that other faculty actually required?

I find myself taking a middle path. I try, on the one hand, not to interrupt, while, on the other, not to allow certain breaches of my personal code. This is a balance between etiquette and pedagogy.

I cannot, for example, tolerate, “Vital signs stable,” a statement meant to mean, “Vital signs were normal.” In fact “stable signs” mean that pulse, blood pressure, respiratory rate and temperature were unchanging. I try to point out that vital signs are most stable for a corpse. I get upset but usually stay silent when arms and legs are referred to as “upper and lower extremities,” but wonder, to myself usually, whether the hand is the “distal upper extremity,” or is this a reference to the “distal phalanx”? Is a finger the “phalanx of the distal upper extremity”?



Is this needless obfuscating jargon necessary? Medicine should be simple and direct and, with the exception of the eponymous disease, it is. However, due to our profession’s history, our simple terminology is in Latin. We have “erythematous” not “red” rashes. How much better it sounds to have “torticollis” than “twisted neck”. What was once clear has become obscure.

My predecessor as editor of this journal, Stan Aronson, once told the story of a chief of pathology at a small community hospital in New York City. The hospital had been recently made into a “teaching hospital,” and the new chiefs of medicine and surgery were certified academicians. A needle biopsy was submitted to the surgical pathology laboratory, and the pathologist sent back the following report: “This is a nasty tumor and I don’t know where it came from.” The new chief of surgery stormed into the pathologist’s office angrily demanding a proper, “academic” report. The pathologist rewrote the report as follows: “Biopsy reveals an anaplastic malignancy of undeterminable origin.” The chief of surgery was satisfied.

Overall, I try to listen and not interrupt when the resident physicians speak. I’m frequently not successful. I hate redundancy, but understand that the student/resident is trying to show that no stone was left unturned in the search for medical answers, whether relevant or not. But I never let “vital signs stable” go unchallenged. I probably will continue to interrupt that phrase until my own vital signs become stable.

– Joseph H. Friedman, MD

A Tale of an Inconsequential Worm

The small English seaport of Yarmouth was not known as an incubator of genius; and certainly the Paget family, Yarmouth brewers by tradition, had sired no great intellects in the recorded past. James Paget was born in 1814, and after a conventional interval of schooling, apprenticed himself to a local practitioner of medicine. By 1834 he realized the utter inadequacy of his formal education and he enrolled then in the medical school affiliated with St. Bartholomew's Hospital [Bart's], in London.

It was during the winter semester of his first medical school year that he entered the autopsy chambers to observe the post mortem examination of a middle-aged barometer-maker who had died after an interval of weight loss, cough and profound fatigue. The autopsy disclosed extensive pulmonary tuberculosis, severe malnutritional changes in the liver and multiple ulcerations involving the lining of the intestines. Paget, standing at the periphery of those in attendance, noted something further: numerous, very small, chalky spots in the muscles of the patient. He borrowed a scalpel and verified the bony texture of these small lesions. The nature of this strange finding [which apparently had produced no symptoms during the life of the patient] puzzled him despite it being called, by his professors, an irrelevant lesion. He then borrowed a hand lens to study them more closely. The hospital owned no microscope so Paget walked to the British Museum where one of the curators directed him to the Botany Department which housed a precious microscope. And after diligent dissection, Paget concluded that the lesion consisted, not of a bony spicule, but of a tiny, calcified cyst and a thread-like worm coiled within it. The pathologist at the medical school verified Paget's findings and gave it the genus name, *Trichina* [from the Greek, meaning hair] and the species name *spiralis*.

Little came of this observation for over a decade. In 1846, Joseph Leidy, a young Philadelphia physician, sat down for his evening dinner. He noticed minute specks of white within his pork chop vaguely reminiscent of the chalky dots that Paget had first described. Leidy later submitted segments of his dinner to analysis and beheld under the microscope the same variety of coiled worms. Still, all of this signified only a curious but symptomless tissue abnormality found occasionally in both man and pig.

It would require another few decades before the full life cycle and clinical implications of this barely visible worm were elucidated. Pathologists were now encountering the chalky white spots in large numbers of human and animal autopsies. Feeding uncooked pork caused the barely visible worms to appear throughout the musculature of experimental animals. But it was not until 1860 that physicians joined these disparate findings into a disease known today as trichinosis. With increasing frequency there arose epidemics involving people sharing one historic feature: They all had eaten inadequately cooked pork [or pork sausage] within days of the onset of abdominal pains, fever, severe muscle aches; and in over ten percent of cases, death. In the German town of Hettstadt, following a banquet of roast pork, 150 took ill and 28 persons died, including the wife of the local butcher who had supplied the meat. Paget's isolated finding was now assuming grave significance.

The full cycle of the trichina worm was finally revealed. When pigs are fed food containing scraps of infected animal flesh they may then become infested with parasitic trichina worms, lodging particularly in their muscles. These small, barely visible worms are actually larva. And when undercooked, parasitized pork is then consumed by a human, the larval forms mature in the human intestine, invade the intestinal walls, mate; and the female then generates thousands of larvae which enter the blood stream of the victim, ending up principally within the skeletal muscles.

Large numbers of humans are thus parasitized by this worm; some surveys place the number between 10 and 15 percent of the meat-eating population. Only when the number of larvae seeded within the muscle is extensive, however, do symptoms arise; and when the infestation is overwhelming, death may ensue.

During the 19th Century, pork meat was the principal source of human trichinosis. Germany was the first nation to forbid the feeding of abattoir scraps to domesticated pigs. They also required that all slaughtered pigs be examined for trichina worms embedded within samples of their muscle. Other nations, including the United States, established similar standards for meat inspection and the incidence of trichinosis has diminished substantially. Curiously, the principal source of clinical trichinosis in this country is now found in those who eat undercooked bear meat or venison.

Leviticus, the third book of the Bible, declares that swine are unclean. "You shall not eat of their flesh or touch their carcasses; they are unclean for you." Some have interpreted this to indicate that the nomadic Israelites possessed considerable knowledge of the life cycles of the parasitic worms. It is more likely that their aversion to the pig was based solely on cultural considerations since many nomadic groups, historically, have avoided eating pork products.

And what about the further career of James Paget, the young medical student who first took the time to examine what others had dismissed as mere specks of bone within skeletal muscle? He graduated from medical school with the highest of academic honors, but because of the poverty within his family was unable to establish a private office or to afford further training. In 1843, he finally secured a position as lecturer in pathology at Bart's, a position that he filled with great distinction. Paget established himself as an astute observer and one of England's most brilliant pathologists particularly in understanding and classifying diseases of bone and joints. He was the first to provide a full description of the bone disorder now called Paget's disease. In later years he was knighted and elevated to the Vice Chancellorship of the University of London as well as the presidency of the Royal College of Surgeons.

Thus was an illustrious medical career launched by a tenacious curiosity about some inconspicuous flecks of chalky material in human muscle. Genius is often little more than a persevering, obstinate resolve to answer life's small questions; or, in Butler's words, "a supreme capacity for taking trouble."

– Stanley M. Aronson, MD

Diabetes Care in the 21st Century
Theory, Practice & Liability

Health Professional Conference

Wednesday, November 10, 1999 – 12:00PM to 6:00PM
Providence Marriott – One Orms Street – Providence, Rhode Island

A continuing education course sponsored by
Brown University School of Medicine & Diabetes Foundation of Rhode Island
Course Director Michael Hein, MD

Program Content

This symposium is designed as a multidisciplinary approach to the imperative of good glucose control, the current social and legal climate surrounding it and the methods by which health care providers can satisfy both masters.

Course Objectives

At the conclusion of this course, participants should be able to:

1. Describe the pharmaco-economic benefits of good blood glucose control in Type 2 Diabetes Mellitus, both for the individual patient, as well as society.
2. Describe the legal pitfalls of not adhering to treatment guidelines promulgated in the face of evidence that good glucose control can delay, diminish, or prevent complications of Diabetes Mellitus
3. Describe ways in which he/she can make changes in his/her mode of practice which would not only reduce liability to the provider, but also enhance the quality of care given to patients.

Who Should Attend

All primary care physicians for adults, specialists caring for adults with diabetes, and non-physician health professionals working with patients with diabetes.

Faculty

James Rosenzweig, MD, Associate Chief of Adult Diabetes Section, Joslin Clinic. Assistant Professor of Medicine, Harvard Medical School, Boston, MA.

Linda Johnson, MPA, Chief Officer of Managed Care Regulations at the Rhode Island Department of Health.

William J. McDonough, MPAH, ARM, FASHRM, Senior Vice President with Health Spectrum at MARSH, Inc.

Education Accreditation

The Brown University School of Medicine designated this educational activity for 4 credit hours in Category 1 towards the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that were actually spent in the educational activity.

Tuition Fees

The tuition fee is $100 for physicians all $45 for allied health professionals. (Interns, residents and fellows: proof of residency from program director is required).

Support for this program is provided by an unrestricted educational grant from SmithKline Beecham.

**Diabetes Foundation Members please deduct 20% of tuition fee*

DIABETES FOUNDATION OF RHODE ISLAND, INC.

IN DEO SPERAMUS

Health Professionals TEAM DIABETES Working Together

Resident Physician Abstracts: Annual Scientific Meeting of the Rhode Island Chapter, American College of Physicians, 1999

INTRODUCTION

The Thirteenth Annual Joint meeting of the Rhode Island Chapter of the American College of Physicians (ACP) was held at the Radisson Hotel on Tuesday, April 6, and Wednesday, April 7, 1999. As usual, participation by medical residents in the Brown University and Boston University-affilaited hospitals was notable for the quantity and quality of the research submissions.

A total of 13 abstracts were accepted for oral presentation and there were over 70 selected for poster presentation. The posters were displayed during the Tuesday evening session in the Plantations Ballroom. Physicians presented their work to their colleagues in a setting reminiscent of a science fair but including food, drink and much discussion. Once again, the posters represented the work of resident physicians and faculty from hospitals in Providence, Rhode Island, including Memorial Hospital of Rhode Island, The Miriam Hospital, Rhode Island Hospital, Roger Williams Medical Center, and the Veterans Administration Medical Center.

Poster presentations remained standing so that participants in the Annual Scientific Meeting could view the posters and continue to interact with the authors on Wednesday, April 7.

As Governor of the ACP, I welcomed the group and the first series of the oral presentations was then made (abstracts included in this issue).

I introduced Dr. Harold Sox, the American College of Physicians representative at our regional meeting. It was especially noteworthy since Dr. Sox was also the President and a Regent of the American College of Physicians/American Society of Internal Medicine. He met many old friends, made many new ones and was an integral participant in our discusison on "Quality and Outcome in the Clinical Practice of Medicine."

Dr. Sox made introductory remarks and a panel discussion revolved around quality and outcome, especially as it pertained to today's health care environment, with managed care as a major player in the arena. Our panelists included: Dr. Peter R. Hoffman, Deputy Medical Director, Harvard Community Health Plan of New England; Dr. Anthony Kazlauskas, Senior Medical Director, United Health Care of New England; Dr. Patricia Nolan, Director, Rhode Island Department of Health; Dr. Edward Westrick, Clinical Coordinator, Rhode Island Quality Partners; Dr. Peter Hollmann, Medical Director, Blue Chip.

At the end of ten-minute presentations by each panelist, there was animated discussion by all participants as well as the audience.

The Irving A. Beck Award was presented to Charles B. Kahn, MD, FACP. This was followd by Meet the Professor Sessions, where experts from the Providence medical community interacted with residents and attending physicians to discuss difficult cases. This year the "Meet the Professor" participants included: Dr. Marilyn Weigner, Dr. Charles Kahn, Dr. Timothy Flanigan, Dr. Lance Dworkin, and Ms. Elaine Narducci, RN.

The afternoon sessions included discussion of the "Hypercoagulable State and Its Therapy" by Dr. Joseph Sweeney and Dr. Anthony Mega. This was followed by further oral presentations.

All present agreed that the meeting was especially successful as it fostered intellectual interaction between students, residents and attending physicians.

–Fred J. Schiffman, MD

Fred J. Schiffman, MD, FACP, is Professor of Medicine at Brown University School of Medicine and Governor, Amrican College of Physicians, Rhode Island Chapter.

Abbreviations Used:

ACP	American College of Physicians
CAD	coronary artery disease
CCU	coronary care unit
CK	creatine kinase
CT	computed tomograph
CXR	chest x-ray
DM	diabetes mellitus
GME	Graduate Medical Education
HIV	human immunodeficiency virus
IBD	inflammatory bowel disease
LDH	lactate dehydrogenase
MAC	*Mycobacterium avium* complex
MI	myocardial infarction
MRI	magnetic resonance imaging
NRMP	National Residency Match Program
NS	not significant
OI	opportunistic infections
PI	protease inhibitors
RWGH	Roger Williams General Hospital
RWMC	Roger Williams Medical Center
tHcy	total homocysteine
TSH	thyroid
USPSTF	United States Preventive Services Task Force

A Novel Method of Assessing Severity of Illness in CCU Compared to Apache Score

Moses Aboagye-Kumi, MD, Kenneth Cristopher, MD, Alanna Coolong, MD, Ara Sadaniantz, MD
The Miriam Hospital

Background

The APACHE score system gives an objective assessment of the severity of illness in the critical care setting. There are certain limitations to this system; e.g., no adjustments made in the presence of hemodynamic support drugs, antipyretic therapy or mechanic ventilation. Critical care patients are subjected to intravenous and intraarterial catheters for medication and monitoring hemodynamics. Furthermore, APACHE score may be cumbersome to evaluate in the Coronary Care Unit (CCU). We hypothesize that the number of catheters would predict outcome measures for CCU patients.

Method

We prospectively collected data during the first 24 hours of admission on 40 consecutive patients to the CCU of the Miriam Hospital betweeen August 18, 1998 and September 2, 1998. Data were collected on the number of catheters inserted, the routine laboratory investigations and the vital signs. The catheters counted included peripheral IV, internal jugular, Swan-Granz catheter, temporary pacemaker, intraaortic balloon pump arterial, endotracheal tubes, and Foley catheters. The length of stay in the CCU and the total hospital stays were also determined. The relationship between length of stay and number of IV medications was compared with the APACHE score and the number of catheters. Relationship between the APACHE score and the number of the catheters was also studied.

Rssults

The number of catheters inserted correlated modestly with CCU length of stay (r=0.47; p=0.02) but not with the total length of stay. APACHE score correlated neither with CCU length of stay (r=0.12; p=1.0) nor the total length of stay. There was no correlation between APACHE score and the number of catheters. We also analyzed the difference in age (70+13 vs. 65+12; p=NS), catheters (3.2+2.0 vs. 2.1+1.8; p=0.07), APACHE score (11.1+2.8 vs. 7.6+2.4; p=0.001), CCU length of stay (3.2+1.2 vs. 3.0+1.4; p=NS), IV medications (3.31+1.8 vs. 2.4+0.8; p=0.04) between women (n=17) and men (n=23).

Conclusion

Counting the number of catheters during the first 24 hours of CCU admission is a very simple tool, which predicts CCU length of stay. This can help manage the CCU, predict hospital course and allocate resources. The significant differences between men and women in our cohort were likely due to the fact that the women were older and their acuity of illness was higher. A larger cohort and adverse outcome tools need to be studied to validate the usefulness of this simple tool. ✧

Diabetes Mellitus in HIV/AIDS Patients Receiving Protease Inhibitors

Omobosola Akinsete, MD, MPH, Rita Jain, MD, Valerie E. Stone, MD, MPH
Memorial Hospital of Rhode Island

Protease Inhibitors (PI) have been hailed as wonder drugs in the treatment of HIV and their use has escalated worldwide as their potent effects have been noted on viral suppression and increased immune function.

However, concerns have been growing over a disturbing but increasing trend of development of metabolic disorders such as lipodystrophy, hyperlipidemia, and diabetes mellitus (DM) which have been reported to be associated with the use of protease inhibitors. The effect of protease inhibitors (PI) on DM is discussed here.

Patient #1 is a 51-year-old Caucasian man diagnosed with HIV/AIDS in 11/90. He presented to the Providence VA Hospital on 3/98 with a 2-week history of vomiting, blurred vision, frequent urination, thirst, generalized weakness, 10 lb. weight loss over the same time frame and 3-week history of sore throat, neck pain, and dry cough. His HIV course had been complicated by HIV-related opportunistic infections - MAC, oral candidiasis and wasting syndrome. There was no past history of diabetes, other endocrinopathies, pancreatitis, alcohol use, and no prior use of pentamidine. Indinavir® had been started 19 months before. Lab values revealed blood sugar of 1116 mg/dl. Based on clinical presentation and laboratory findings, DM was diagnosed. Patient was started on insulin, but DM has since been difficult to control and doses of insulin have had to be adjusted frequently.

Patient #2 is a 34 year-old Hispanic female with HIV/AIDS diagnosed in 9/94 after an episode of PCP. She also has a history of Porphyria Cutanea Tarda, Hepatitis C and other HIV-related opportunistic infections including perianal herpes simplex and oral hairy leukoplakia. She developed DM secondary to pancreatic injury from pentamidine, which was well controlled with Glipizide®. She was started on Saquinavir® HCG in 3/96 after persistent high viral loads; this was changed to Ritonavir® one month later due to its potentially greater clinical response. Over the next eleven months, blood glucose became difficult to control. Varying between 205 and 389 mg/dl and Glipizide® had to be adjusted frequently.

These cases demonstrate the two types of DM-related complications of PI treatment: 1). Development of new DM 2) Worsening of known DM. In both cases, patients' blood glucose becomes more difficult to control in the absence of systemic infections and ketosis after beginning therapy with PIs. However it needs to be emphasized that reported metabolic and endocrinology effects of protease inhibitors are still controversial, but if proven, could lead to contraindications to or noncompliance with therapy and influence the way we treat HIV. ✧

Unusual Presentation of Enteropathic Arthritis

Tanya Ali, MD (Associate), Bernard Zimmerman, MD (FACP)
Roger Williams Medical Center, Boston University

Purpose

Peripheral arthritis occurs in approximately 10-20% of patients with inflammatory bowel disease (IBD). Arthritis is most commonly oligoarticular, asymmetric and predominantly involves the joints of the lower extremity. Residual deformity and radiologic evidence of bony destruction are rare. The current study was designed to investigate unusual patterns of arthritis associated with IBD.

Methods

Seven patients with h/o both IBD and arthritis were identified from the consultation files of the rheumatology and gastroenterology services at RWMC and PVAMC. Patterns of arthritis, other relevant clinical features and pertinent laboratory findings were noted on chart review.

Results

Of the 7 patients, one had unusual pattern of arthritis. This 22-year old, white male with Crohn's, presented with symmetrical arthritis of DIP, PIP, MCP joints with significant flexion deformity. Erosive changes of MCP joints were noted on x-ray. The patient had flare up of his peripheral arthritis with each episode of active bowel disease. Rheumatoid factor and ANA were negative.

The remaining 6 patients developed arthritis with or after the onset of IBD. Typical patterns of joint involvement were found to be oligoarticular and asymmetrical. Commonly affected joints were knee, ankle, wrist, elbow and small joints of the hand. No residual deformity or radiological evidence of joint destruction were present in these 6 cases.

Conclusion

Our study revealed that symmetric arthritis, joint deformity and radiologic evidence of joint destruction although uncommon, can occur in patients with IBD. The differential diagnosis of symmetric erosive arthritis should also include enteropathic arthritis. ✧

Ceramide Provides an Apoptotic "Death Signal" Which Potentiates the Anti-Tumor Effect of Taxol

Dilshad Blackinton, MD, Shashi Mehta, MD, Harold Wanebo, MD
Roger Williams Medical Center

Introduction

Ceramide, a sphingomyelin metabolite, provides the "death signal" for apoptosis in mammalian cells. Paclitaxel, an active chemotherapeutic agent, blocks cells in the G2/M phase of the cell cycle. We explored the interaction of paclitaxel and ceramide in a variety of solid tumor cancer cell lines to determine whether there was synergism in inducing cytotoxicity and apoptosis.

Purpose

To determine whether the anticytoxic effect of taxol may be enhanced in the presence of ceramide in human tumor cell lines.

Methods

The effects of paclitaxel and ceramide on (1) cell growth of Tu138 was evaluated by MTT dye assay, (2) cell cycle progression by flow cytometry, (3) cell apoptosis by TUNEL assay and (4) tumor growth and weight in nude mice. To corroborate in vitro findings, Tu138 cells were planted subcutaneously on nude mice. Treatment with paclitaxel (120 ug/0.1 mL) + ceramide (500 ug/0.2mL) was begun on day 4 with thrice weekly injections administered subcutaneously near the tumor site for 5 weeks.

Results

Cell growth studies showed synergistic growth inhibition with paclitaxel + ceramide and was confirmed by a 50% isobologram analysis in six separate experiments (p value<0.05). The TUNEL assay also confirmed enhanced apoptosis by the combined treatments. Cell cycle progression studies suggested that the target population of cells was either in the S or G2-M phase of the cell cycle. Preliminary in vivo studies showed equivalent growth of Tu138 cell-tumor in control and paclitaxel treated mice whereas the ceramide injected group showed a slight decrease in tumor size at week 5. The paclitaxel + ceramide group showed a decline of 70% in tumor size compared to control group. There was also significant reduction in tumor weight (88% in paclitaxel +ceramide group vs. control).

Conclusion

Ceramide enhances paclitaxel-mediated cytotosicity in several tumor cell lines and augments the anti-tumor effect of paclitaxel on one of the lines (TU 138) in an in vivo nude mouse model. The mechanism is not yet defined, although it may be related to changes in intracellular ceramide and Fas ligand mediated events. ✧

Presentation of a Patient with Presumed Listeria Rhomboencephalitis

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Brown University Department of Internal Medicine, The Miriam Hospital

A 64 year-old right-handed man presented with 30 days of progressive generalized weakness, and five days of right facial and tongue numbness. He also developed a temperature to 38.3 C two days prior to admission. Of note, he had lived in El Paso and made frequent trips to Mexico in the 1960s. Past medical history included coronary artery disease and a seizure disorder. Medications on admission included: Tegretol®, Procardia®, Prinivil® and Lasix®.

Physical examination on admission was significant for a temperature of 37.8 C, a 2 of 6 systolic murmur, diplopia with right gaze and decreased facial sensation.

Laboratory data on admission included a white blood cell count of 9300 cells per microliter with a normal differential. Lumbar puncture revealed 79 nucleated cells per microliter with 60% neutrophils and 30% lymphocytes. Cerebrospinal fluid protein was elevated at 122 milligrams per deciliter, and glucose was normal. Magnetic resonance imaging of the brain showed a calcified lesion in the right frontal lobe and a cystic lesion with surrounding edema in the right brain stem.

The patient was started on ceftriazone, steroids and albendazole with a working diagnosis of neurocysticerosis. His diplopia progressed and he developed refractory hiccoughs. On hospital day 4 the patient reported worsening facial numbness and ataxia of his upper extremities. He then underwent a repeat MRI, which showed progression of the lesions which now included the pons and middle cerebellar peduncle, consistent with brainstem encephalitis. The patient was started on ampicillin and gentamicin, while the steroids and albendazole were discontinued. That evening he became lethargic, then had a cardiac arrest and died. Listeria rhomboencephalitis is the presumed diagnosis in this case. Full autopsy results to confirm the diagnosis are pending at this time.

In conclusion, brainstem encephalitis can present with non-specific symptoms. It is often due to *Listeria monocytogenes.* Physicians should be aware of this lethal, but easily treatable entity. ✧

Two Cases of Hepatic Hydrothroax Without Ascites

Alice Fan, MD, Michael Vecchione, MD, Allan Erickson, MD
Brown University School of Medicine, VA Medical Center

Described are two cases of right-sided transudative pleural effusions secondary to cirrhosis. First, a 44 year-old gentleman with a history of hepatitis B and C, alcohol abuse, and cirrhosis presented with chest pain and shortness of breath. Physical exam revealed decreased breath sounds over the lower 2/3 of his right lung field; his left lung was clear. CXR confirmed a large pleural effusion while an abdominal ultrasound revealed no significant ascites. A diagnostic and therapeutic thoracentesis was performed: fluid analysis was consistent with a transudate. Gram stain, culture and cytology were negative. Chest CT showed no mass lesions or other pathology. Endocardiogram showed normal left ventricular function. Patient had significant symptomatic improvement and was discharged home on diuretics.

Our second case was a 69 year-old gentleman with a history of hepatitis C, alcohol abuse, and cirrhosis with portal hypertension who presented with diarrhea and vomiting. During the course of his admission he developed shortness of breath. Lung exam was consistent with a large right pleural effusion, confirmed by CXR. He did not have significant ascites on physical exam or by abdominal ultrasound. He had no evidence for an underlying pulmonary infection. The pleural fluid was found to be a transudate. Gram stain and culture were negative. Echocardiogram showed normal left ventricular function. He also underwent therapeutic thoracentesis and further diuresis with resolution of his dyspena. Although his pleural effusion had not completely resolved, he was clinically stable at the time of discharge.

Hepatic hydrothorax is a pleural effusion that occurs in patients with cirrhosis; peritoneal ascites accumulates in the pleural cavity by leakage through diaphragmatic defects. It is postulated that negative pressure in the thoracic cavity causes ascitic fluid to enter the pleural cavity and that the same negative pressure gradient assures unidirectional flow. It is rare for this process to occur without significant ascites present in the abdomen. The diagnosis is suggested when cardiopulmonary disease is excluded in a patient with a transudative pleural effusion and cirrhosis. Treatment remains the same as for hydrothorax with ascites: diuresis with therapeutic thoracentesis as a temporizing measure to relieve severe dyspena. The diaphragmatic defects may close spontaneously, but more likely these effusions will require chronic management. ✧

Serum Cystatin C is a Major Independent Determinant of Fasting Plasma Total Homocysteine Levels in Coronary Artery Disease Patients with a Normal Serum Creatinine

Gintaras Liaugaudas, MD, Linda Bausserman, PhD, Omobosola Akinsete, MD, Andrew G. Bostom, MD
Brown University School of Medicine

Serum creatinine, a surrogate for both renal function and homocysteine generation, is a determinant of fasting plasma total homocysteine (tHcy) levels in coronary artery disease (CAD) patients. We examined whether serum cystatin C, a more sensitive indicator of glomerular filtration rate, would better predict fasting tHcy levels among stable CAD patients with normal creatinine levels. Fasting plasma tHcy, folate, vitamin B12, and pyridoxal 5'-phosphate levels, along with serum cystatin C, creatinine, and albumin levels, were determined in 164 consecutive CAD patients (mean age 61 ± 9 years; 78.7% men) with stable CAD, and a serum creatinine ≤ 1.4 mg/dL. Multivariable linear modeling revealed that serum cystatin C was independently predictive (partial $r=0.379$; $p<0.001$) of fasting tHcy levels after adjustment for age, sex, vitamin status, albumin, and creatinine levels. Moreover, cystatin C alone determined more than half of the variability in tHcy (i.e., R2) accounted for by the full model. In contrast, creatinine levels were not independently predictive of fasting tHcy levels in this model ($o>0.2$), or an identical model which excluded cystatin C ($p>0.2$).

Serum cystatin C levels may reflect subtle decreases in renal function that independently predict fasting tHcy levels among CAD patients with a normal serum creatinine. ✧

Communication During the NRMP Match: Insight From Internal Medicine Program Directors

Laura Obbard, MD (Associate), Dominick Tammaro, MD, Michele Cyr, MD
Rhode Island Hospital

Many fourth year medical students write letters, call, or go on second look visits to residency programs during the NRMP match. While it is known that these informal interactions occur, it is unknown how they are perceived by Program Directors, or if the Program Directors' opinions vary depending on their type of program (University or Community) or previous match rate (based on 1997 and 1998 GME data).

In order to evaluate this communication, a questionnaire was sent to 202 randomly selected Internal Medicine Program Directors (43% University, 57% Community; 74% completely matched both 1997 and 1998, and 26% unmatched in 1997 and/or 1998). One hundred and twenty -three surveys were returned (61% response rate). Of those programs responding, 43% were from University programs, 57% Community, 75% completely matched 1997 and 1998, and 25% were unmatched in 1997 and/or 1998. Program Directors graded 15 areas of communication on a 5-point scale, stating whether they added to or detracted from the student's application.

Program directors felt that 11 items added to the application. They were (in order of most important to least important): coming for a second look visit; hearing from a colleague at another program about the student; making specific comments about the interview day; writing a thank you letter; sending additional information (when appropriate); asking additional questions about the program; stating they will rank the program #1 or very highly; corresponding with e-mail or phone calls; and writing within 10 days. Program directors felt that four items detracted from the application (from most detracting to least detracting): questioning rank at a program, sending the same letter to multiple interviewers, not mentioning how they plan to rank a program, and not writing a thank you letter.

Community programs were more likely than University programs to feel positively when students stated that they would rank a program highly or at the top ($p<0.5$). Unmatched programs were more likely than matched programs to feel that coming for a second look ($p<0.2$) and writing a thank you letter ($p<0.5$) adds to the application.

These data confirm that some types of communication are viewed favorably by Program Directors during the match process. Whether this communication actually influences rank lists is unknown. ✧

Outcome Predictors of Abnormal Thyroid Function and Morphology in a Community Screening Program

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Department of Medicine, Division of Endocrinology, Brown University School of Medicine, Rhode Island Hospital

BACKGROUND

An estimated 11 million Americans have thyroid dysfunction and less than one half are aware of it. There is no clear cost-effective method for screening communities; therefore, casefinding in the medical setting and in high risk populations is pursued.

PURPOSE

To determine the frequency of detectable thyroid function test and exam abnormalities in an ambulatory cross section of the general population and seek correlation of symptoms or historical characteristics of the population that predict abnormal thyroid function and morphology.

METHODS

Through media advertisement participants were invited to receive a sensitive TSH test and a subpopulation was offered an exam of the thyroid gland by an experienced examiner. The screening was conducted in June 1995 at the Rhode Island Hospital and the Roger Williams Medical Center. Respondents completed a questionnaire of demographic data and 34 questions regarding symptoms suggestive of thyroid disease. Univariate chi-square analysis was performed on 34 independent variables against the outcome variables: elevated TSH, suppressed TSH, presence of a goiter and an abnormal size thyroid gland.

RESULTS

Demographic data previously reported, Twigg et al, RI ACP, 1997. Of the historical factors and symptoms surveyed, 5 variables in the univariate analysis were significant predictors of an elevated TSH by chi-square analysis. Nervousness had a significant correlation with predicting an abnormal size thyroid gland and the history of a thyroid nodule was significant in association with a goiter. A low TSH had 13 variables that had significant associations i.e., increase in frequency of bowel movements, a decrease in appetite and nervousness. Logistic regression models for predicting outcome indicated associations between an abnormal size thyroid with a family history of diabetes and the subjective complaint of nervousness; associations with a goiter on exam were family history of diabetes and lupus as well as nervousness. An association between an elevated TSH and a previous history of thyroid treatment, a family history of diabetes and an increased appetite was noted.

CONCLUSIONS

It is possible to conduct a community screening program and provide an exam of the thyroid without great expense to the patient. Several historical factors predicted abnormal TSH and exam outcomes. Albeit potentially different from the general population, ours was self-motivated to seek medical care, concerned about their thyroid and may represent the population that would present to a general Internist. ✧

Human Granulocytic Ehrlichiosis

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Brown University School of Medicine

A 32 year-old HIV+ woman presented in clinic with fever of 102° F, headaches, and neck stiffness since that morning. She awakened with these symptoms which progressively worsened during the day. In addition, the patient reported lethargy (rendering her bedridden), retro-orbital pain, non-productive cough, and loose foul smelling stool. She stated she had been visiting her mother, who was recently hospitalized at Kent County Hospital for pneumonia and cardiac disease. She denied any other sick contacts. Furthermore, she reported a tick bite approximately 3 weeks prior to admission and had since been in the wooded areas of Connecticut. Past medical history was significant for HIV, diagnosed in 1993 with her most recent CD4 count being .656 K/u L and viral load 1683 copies/mL. She was not on antiretroviral therapy. She was hepatits C+ and reports chronic sinusitis. Physical examination demonstrated a temperature of 102° F, frontal and maxillary tenderness, and profound neck stiffness with limited range of motion. The white blood cell count was 5.3 x 1000/cu mm, the hematocrit was 40.4%. the hemaglobin was 14.2 gm/dL, and the platelets were 188,000/cu mm. The differential showed 91% neutrophils, 2% bands, 5% lymphocytes, and 2% monocytes. Liver transaminases were normal. The peripheral blood smear revealed Dohle bodies. The cerebral spinal fluid (CSF) was clear with 14/cu mm nucleated cells, no neutrophils and 94% lymphocytes. The CSF protein was 34 mg/dl and glucose was 55 mg/dl. Her chest x-ray was negative for infiltrates and sinus films showed left maxillary sinus thickening. The patient was admitted for bacterial meningitis and placed on ceftriaxone. On day 2 of hospitalization, she was diaphoretic and tachycardic, with a temperature of 105° F. A head CT was obtained which revealed an acute sinusitis superimposed on a chronic sinusitis. Antibiotic was switched to cefuroxime. On day 3, she developed myalgias and continued to have a temperature of 104° F. Her platelets dropped to 62,000/cu mm and AST was 84 IU/L. On day 4, her platelets were 30,000/cu mm and white blood cell count 2.4 x 1000/cu mm. On day 5, the platelet count was 32,000/cu mm and white blood cell count 2.1 x 1000/cu mm. On day 6, her platelet count was 45,000/cu mm, white blood cell count 3.5 x 1000/cu mm, AST 109 IU/L, and ALT 64 IU/L. The patient was placed on doxycycline and serum was sent for PCR for human granulocytic ehrlochiosis, which was positive.

In patients who present with sudden febrile influenza-like illness following outdoor activities, HGE should be suspected. Important clues to the diagnosis are multisystem involvement without specific localizing signs and symptoms. Infected patients characteristically complain of headaches, myalgias, shaking chills, and fever. Thrombocytopenia, leukopenia, and elevated hepatic transaminases are common laboratory findings. Occasionally, neutrophilic morulae are demonstrated on the peripheral smear. A high percentage of *Ixodes dammini scapularis* ticks in Connecticut were shown to contain HGE agent DNA. Therefore, this entity is of particular importance in our geographic area. ✧

Localized Amyloidosis Presenting as Pelvic Pathology

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Brown University Residency Programs in Internal Medicine

A 53 year-old African American female presented with early satiety, abdominal cramping, fatigue, and a 25 lb. weight loss over a five month period. Other relevant review of systems was negative. Family history was significant for liver and lung cancer but was negative for breast and ovarian carcinoma. Physical exam, including a gynecologic exam, was unremarkable. These symptoms prompted a CT scan of the abdomen and pelvis that demonstrated omental caking highly suspicious for ovarian carcinoma. Supporting this impression, an explanatory laparotomy revealed diffuse tumor studding the peritoneum, omentum, and both ovaries. Finally a CA 125 was found to be high at 79 U/ml, and the oncology service was asked to advise on treatment of ovarian carcinoma.

Surprisingly, on frozen section the tumors consisted of waxy, eosinophilic fibrils that, when stained with Congo red and viewed under polarized light, had the characteristic apple-green fluorescence of amyloid protein. In order to find the source of this amyloid deposition a serum protein eletrophoresis was performed, which was positive for an IgG-lambda spike. A subsequent bone marrow biopsy confirmed the diagnosis of multiple myeloma. Thus far, the patient has not developed symptoms of systemic amyloidosis and is being treated for her underlying disease.

Amyloidosis is a condition in which protein with a ß-pleated sheet structure accumulates either systematically or in discretely localized masses. The disease is divided into two subsets based on this protein's composition: 'Immunicyte derived,' where deposits are immunoglobin light chains (Amyloid L) and 'non-immunocyte derived' with an electrophoretically distinct protein (Amyloid A). Localized amyloidosis (also known as tumoral amyloidosis) is usually associated with the AL protein type. Although tumoral amyloidosis has been described in many anatomic sites, this case of localized amyloidosis presenting as pelvic pathology is quite unusual, and has not been described previously in the medical literature. This case also illustrates an important point about the CA 125 laboratory test. Since CA 125 is present in endometrial tissue and in serious and mucinous uterine fluids, any process involving these tissues can result in a high CA 125. This case serves as a reminder that the CA 125 is not useful in establishing the diagnosis of ovarian carcinoma, especially when there is evidence of widespread pelvic disease. ✧

Evaluation of a Woman's Preventative Medicine Program in a Residents' Clinic

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Roger Williams Medical Center

Background

Medical authorities have established guidelines for preventive medicine programs in women. We reviewed the charts of 148 women over the age of 40 being followed in the residents' clinic of Roger Williams General Hospital (RWGH) to determine how many adhered to the guidelines of the U.S. Preventive Services Task Force (USPSTF) Guide to Clinical Preventive Services: Blood pressure measurements each visit, Pap smear every year, occult blood cards and/or signoidoscopy after the age or 49, mammography every year after 49, total blood cholesterol between age 45 and 64, assessment for a drinking problem, counseling for adequate calcium intake and vitamin D as well as for exercise encouragement, and discussion of hormone replacement therapy with perimenopausal women.

Purpose

To determine how many patients were being treated in accordance with the USPSTF guidelines.

Method

We reviewed the charts of all women over the age of forty being followed in our residents' clinic. Of the 148 women, 123 were over the age of 50.

Results

73.64% of the patients were assessed for hypercholesterolemia, 97.29% for hypertension, 46.34% for colorectal cancer, 44.59% for cervical cancer, 76.79% for breast cancer. Also 8.78% patients were advised to take calcium, 5.40% to take vitamin D and 14.68% to exercise as a preventive measure. Hormonal replacement therapy was discussed with 3.3.7% of the patients.

Conclusion

Nearly all patients were screened for hypertension. Over 75% were screened for breast cancer with mammograms. However, in less than 50% of patients were all guidelines being adhered to. And less than 10% were being counseled regarding hormone replacement therapy, exercise and prevention of osteoporosis.

Recommendation

We will implement a program to encourage the residents to adhere to the UPSTF guidelines by teaching them in a series of noon-time conferences about the subject and placing posters in every examination room available and teaching the patients by offering to let them watch a video of what they should expect from us, while they sit in the waiting room. Then, after 6 months, we will re-review the 148 charts to assess the improvement of our residents' practice. ✧

Lactate Dehydrogenase and Isoenzymes in Myocardial Infarction: a One-Year Retrospective Study

John S. Straus, MD (Associate), Mark Braun, MD
Roger Williams Medical Center.

Background

Guidelines endorsed by the ACP, American Heart Association, and the American College of Cardiology recommend that levels of lactate dehydrogenase (LDH) and LDH isoenzymes be tested only in patients admitted with negative creatine kinase (CK) enzymes and more than 24 hours of chest pain (J Am Coll Cardi*ol* 1990;16:249-92). A recent utilization review and national survey found that LDH measurement did not add clinically useful data and result of a phone survey of 100 hospitals showed that 66% still used LDH isoenzymes in every patient (*Arch Intern Med* 1997;157:1441-4). At the Roger Williams Medical Center (RWMC) total LDH is part of the ER cardiac panel and isoenzymes are ordered fairly routinely on MI admissions.

Methods

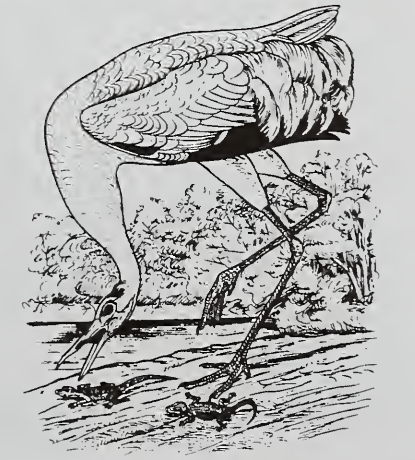
The charts of all patients discharged from RWMC with a final diagnosis of MI, or that died in the hospital due to a MI, between 4.1.97 and 3.30.98 were reviewed for total CK, CK-MB fraction, total LDH, and LSH isoenzymes.

Results

During the year studied there were 155 patients who died of or sustained an MI. Of the 149 charts available for review only three had a negative total CK or CK-MB fraction but with positive total LDH and isoenzymes, even after serial testing in many instances. Total LDH was not done on 6 patients and isoenzymes were not done of 45 patients. Thus CPK was diagnostic in 98% of the 149 patients tested and LDH was only diagnostic in 22% of the 104 patients tested with a final diagnosis of MI.

Conclusions

The total LDH and LDH isoenzymes were rarely positive in the presence of negative CK and CK-MB fractions and in only one patient was it essential in establishing the diagnosis of MI, although knowledge of this did not influence treatment upon chart review. At the RWMC, testing for LDH isoenzymes is done once a day (in the afternoon) but may only be done every other day and not at all on weekends, thus minimizing the usefulness of testing for isoenzymes in the acute setting. Data at RWMC confirm prior studies and support following established guidelines, or eliminating LDH testing altogether, especially in light of mounting acceptance of cardiac Troponin I as both an early and late marker of myocardial injury. ✧



Crash and Learn: 21st Century Interdiciplinary Medical Approach to Injuries and Biomechanical Trauma

Thomas F. Morgan, MD

In the area of posttraumatic syndromes and their causation mechanisms, doctors can use a mechanistic approach which can improve patient care, patient satisfaction and simultaneously promote injury prevention. Further, this user-friendly medical science methodology can clinically correlate injuries and trauma that will render diagnoses, causation, injury severity, prognosis and impairment assessments. The following case presentation will illustrate these points.

MEDICAL INJURY EVALUATION

Trauma Scene

At 6 p.m. on 7/18/90, in heavy traffic, at a major intersection in Warwick, RI, a 28-year-old restrained female driver of a 1990 Acura Legend was struck on the driver's side door by a 1989 Ford Step Van. This impact caused the Acura to move and rotate 90 degrees clockwise. The police were called. The patient was initially unconscious and bleeding on the left side of her face.

Injury/Post-Injury

She was taken to the Emergency Room, where she complained of a headache. The patient was alert and had approximately 30 minutes of amnesia. She was admitted to the hospital with a laceration on the left side of her face, left periorbital edema, bruising and a small laceration of the left upper arm and shoulder region. A CT scan of the head revealed a hemorrhagic contusion of the left frontal, left temporal area and an old lacunar infarct in the deep right frontal lobe. There was also a tripod fracture of the left zygoma involving the zygomatic arch and lateral wall of the left orbit. There was some bony displacement. The patient was initially felt to have brain swelling with a mild shift from left to right and was treated emergently with dexamethazone, mannitol, phenytoin and phenobarbital. The patient also had a plastic surgery evaluation for repair of the zygomatic arch and tripod fracture. The patient had a dental evaluation for fracture of her left upper teeth. A repeat CT scan showed resolving contusions. An EEG revealed bilateral theta slowing. The patient was discharged in 10 days in an improved condition.

On 8/6/90, approximately three weeks after her accident, the patient was seen in the Neurology office. Patient gave a history that she was hit broadside in a motor vehicle accident and struck the left side of her face and left elbow and lost consciousness. The patient's major complaints at that time were trouble with word finding, emotional lability, easy crying and headache. She had a history of anticardiolipin clotting disorder and a resolved cerebrovascular accident approximately five years previously.

The injury syndrome diagnoses:

1. Brain concussion.
2. Left frontal and left temporal hemorrhagic contusion.
3. Tripod fracture of the left zygoma.
4. Laceration and bruising to the left periorbital region.
5. Bruising and minor laceration to the left shoulder blade, left upper arm and left elbow.

Anatomic injury points:

1. Left side of forehead and scalp, left frontal and temporal brain cortex.
2. Left periorbital and facial bruising along with a tripod fracture of the left zygoma.
3. Bruising and minor laceration to the left shoulder, shoulder blade, left upper arm and elbow.

Abbreviations Used:

AIS	Abbreviated Injury Scale
CT	computed tomography

TRAUMA CAUSATION EVALUATION

This trauma scenario was a driver's side lateral impact with a mismatched large size truck striking the driver's side door and front panel of a midsize Acura. This caused deployment of airbags with a restrained driver. The principle direction of trauma was primarily to the left head, brain, face and left shoulder region. This impact caused initial body motion to the left toward the impacting truck followed by a rebound motion to the right.[11] The predicted trauma points of injury were to the head, brain cortex, left facial region, left shoulder and upper extremity. Trauma variables that affect injury risks included the mismatch of truck to car and the lateral impact forces along with translational and rotational accelerations to the brain.[6]

MEDICAL OPINION

The patient sustained cognitive and behavioral changes related to frontal and temporal lobe contusions along with posttraumatic anxiety, depression and migraine headaches. The subsequent course for this patient required a cognitive and behavioral treatment program as well as psychiatric intervention for anxiety and depression. In addition, the patient required treatment for post-traumatic headache of the migraine type.

The injuries clinically correlated with the traumatic mechanism of injury and the potential points of injury from the trauma.

Subsequently, the patient sustained posttraumatic cognitive and behavioral change related to frontal temporal lobe contusion along with posttraumatic anxiety, depression and migraines. The initial injuries would be classified by the Abbreviated Injury Scale (AIS II moderate and AIS III serious) for brain concussion and brain contusion respectively.[7] Skin laceration and bruising would be classified as Abbreviated Injury Scale I and considered minor.

CASE DISCUSSION

This case demonstrates the clinical usefulness of a mechanistic approach to the patient's posttraumatic complaints.[1] It is important to realize that injuries are not caused by disease but rather by trauma. Therefore, the treating physician must understand some of the basics of the trauma mechanisms of injury. In my experience, this has improved patient care, patient satisfaction and injury prevention. Using a mechanistic approach often leads to improved diagnoses and better anatomical localization of injuries. This approach frequently identifies injuries that have been overlooked or misperceived. Additionally, many times patients have a preexistent disease process such as a degenerative disease that can look like an acute injury. Thus, understanding the mechanism of injury will help with the differential diagnosis.

If one has a knowledge of the trauma variables, this will promote the opportunity to talk about increased or decreased injury risks and injury prevention (i.e., wearing seatbelts, proper adjustments of head restraints, etc.). From a physician's point of view, a user-friendly mechanistic approach will provide more education to both doctors and patients with posttraumatic syndromes. This understanding can also be cost-effective. Knowledge of the mechanism of injury will reduce future diagnostic tests that are unnecessary and also allow the treating physicians to intervene and avoid unnecessary extensive chiropractic or physical therapy treatments.

Finally, understanding the trauma mechanism of injury can be time-efficient in a medical office setting. Completing simple questionnaires about the accident can serve to educate doctor and patient on mechanisms and outcomes. These can allay patient fears regarding posttraumatic complaints and in many instances decrease return calls to the office as well as post-accident Emergency Room visits.

CONCLUSION

It is my opinion that this interdisciplinary mechanistic approach will improve care, better-educate patients and provide the potential clinical research in the area of posttraumatic syndromes and motor vehicle accidents. This process can quantitate impacts as well as posttraumatic injuries and their outcomes and will make our clinical case experience more scientific.[10]

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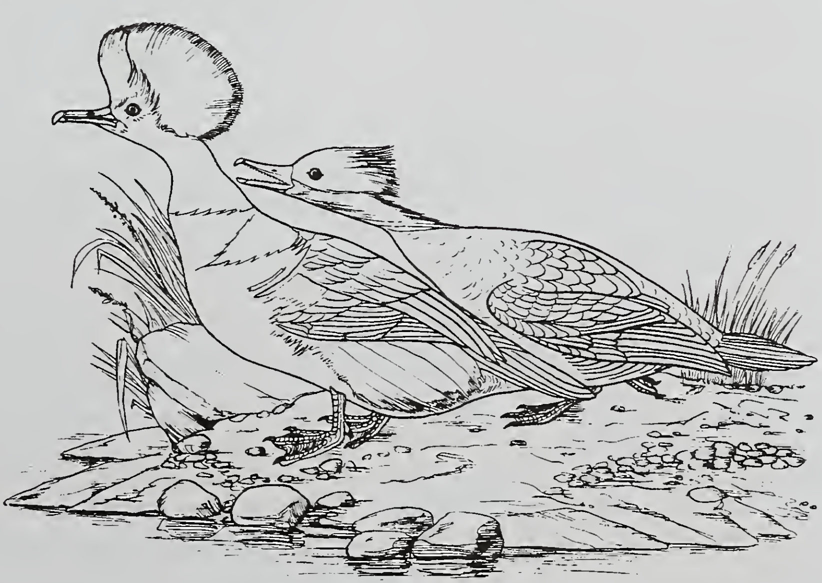

Supracervical Abdominal Hysterectomy: A Technique Whose Time Has Come (Again!), Including an Opinion by the Late Dr. David Nichols

Robert R. Williams, MD

When I was a resident at the Providence Lying-in Hospital in the early 1970s, I did a rotation in gynecological surgery with the late Dr. Henry McDuff at Rhode Island Hospital. Dr. Mc Duff told me that in 1972 the hospital had had an accreditation evaluation. Evidently surgeons at Rhode Island Hospital had performed a series of 400 consecutive hysterectomies without a single supracervical hysterectomy. The evaluator had asked Dr. McDuff, "Do all of your gyn surgeons have such good technique, or such bad judgment that there wasn't an indication for even one supracervical hysterectomy?"

At Women & Infants' Hospital of Rhode Island, my experience is that little has changed in the past twenty-five years regarding this issue. Supracervical hysterectomies remain uncommon in situations where they might be appropriate medically, and they are still rare as an elective procedure.

Anila Verma, MBBS, PhD, et al, in a 1999 review of statistics regarding surgical approach to hysterectomies performed in Rhode Island for the previous five years didn't even include supracervical hysterectomy as an option! The cases performed in 1998 at Women & Infants' Hospital are divided between total abdominal hysterectomy (63%), vaginal hysterectomy (24 %), and laparoscopically assisted vaginal hysterectomy (13%). [1]

Other parts of the country have also been slow to re-embrace the concept of performing supracervical hysterectomy, but some, at least, are ahead of Rhode Island. ES Sills, et al, in their May 1999 article state: "While the TAH (total abdominal hysterectomy) rate declined in New York State, from 34.0 in 1990 to 28.4 in 1996 (rate being the number of procedures per 10,000 females of New York State), the SCH (supracervcial abdominal hysterectomy) rate increased nearly five-fold during the same period, from 0.62 to 3.07."[2] *Thus, in New York State in 1996, there was one SCH performed for every ten TAHs!*

As D.E. Darnell Jones, MD, et al state in their recent article: "There is some persistence in the belief that 'real gynecologicsts' always remove the cervix."[3]

For years I have had a sneaking suspicion that I was not doing some of my patients a favor by removing their cervices during hysterectomy operations for benign disease. We don't routinely remove the appendix during these cases, and the appendix is much more likely to cause the patient problems in the future! So why remove the cervix? Is it because the cervix is a useless organ, except for childbirth? I intuitively doubt that! The cervix is the center of all of the supporting tissues at the base of the pelvis—the hub uniting and supporting the uterosacral ligaments, the cardinal ligaments, and the endopelvic fascia. It cannot possibly be true that if we tack the tip of the uterosacral pedicle (which will be necrosing in a few days) to the side of the vaginal apex with some absorbable suture, that we will maintain just as much support for the vagina as if we left the cervix in place.

> *Supracervical hysterectomies remain uncommon in situations where they might be appropriate medically ...*

Recently I was forced to act on my belief that elective abdominal supracervical hysterectomy is indeed an excellent technique to employ in selected patients. A patient came to me whom I felt was the perfect candidate for such a procedure, and she specifically requested that her cervix be preserved. She is a 44 year old nulligravid woman, who did not want to have children, and who was suffering from heavy, painful periods due to her 14 weeks gestation-sized leiomyomatous uterus. She had had regular annual Pap smears, which had always been normal. She is a non-smoker. Her vagina, in my opinion, would not have allowed me to perform an LAVH (laparoscopically assisted vaginal hysterectomy) without significant angst to myself and risk to the patient (even if we had used Depo-Lupron preoperatively to reducethe size of her fibroids). She had read articles in women's journals about beneficial effects of maintaining the cervix to enhance pleasure during sexual intercourse. I performed an elective supracervical hysterectomy on this patient.

Abbreviations Used:

LAVH	laparoscopically assisted vaginal hysterectomy
SCH	supracervcial abdominal hysterectomy
TAH	total abdominal hysterectomy

I have practiced at Women & Infants' Hospital of Rhode Island — a bastion of New England conservatism — for approximately twenty five years, and I had never seen or heard of this operation being performed at our institution before on an elective basis. The case went extremely well, and took a great deal less time than I had expected (60 minutes with a third year resident first assisting, compared to my average 90 to 120 minutes for a TAH). I was feeling elated about the case when I returned to the locker room to change, and found none other than Dr. David Nichols- - the former Chief of Ob-Gyn at Women & Infants' Hospital and Professor of Ob-Gyn at Brown University. He was also an internationally known authority on hysterectomy techniques. Initially I was hesitant to discuss the case with him — afraid that he would give me many good reasons why I should never perform another elective abdominal supracervical hysterectomy! Finally, I built up my courage and "confessed" to Dr. Nichols that I had just performed an elective supracervical hysterectomy at my patient's request.

I then rushed on to enumerate the advantages of this technique (Table I)

I hoped that Dr. Nichols' reprimand would not be too severe. To my great relief Dr. Nichols said that he agreed with me — and that we should be doing a lot more supracervical hysterectomies. He said that they were done routinely back in the 1920s when the risks of anesthesia were much greater and doctors wanted to keep the surgery as brief as possible. Then, in Baltimore in the 1930s they found that 5% of patients with cervical cancer had previously had supracervical hysterectomies. Thus supra-cervical hysterectomy fell out of favor completely. However, he said doctors did not have Pap smears in the 1930s, or colposcopy. Dr. Nichols' main concern in this conversation was that if we only did supracervical hysterectomies, then the residents would never learn how to do total abdominal hysterectomies.

Since I performed my first elective supracervical hysterectomy on May 19, 1998, I have performed a total of seven. I have been extremely pleased with this procedure. I have used the standard procedure described in texts.[5] The hysterectomy proceeds as usual until the uterine vessels have been ligated. I then take one small bite of the upper cardinal ligament (leaving most of this ligament intact) and this small bite is ligated on each side. The uterus is then excised from the cervix at the level of the internal os, using a hot cautery electrode, and beveling the cervix so as to excise the upper endocervix, but without reaching the uterine vessels. I then use a leep loop to shave off another 1/2 to 1 centimeter of the upper endocervical canal, to ensure that there is no endometrial tissue left, and then sew the beveled edges of the cervix together with 0 Dexon figure of 8 sutures, sutured in the A-P plane. (Table 2.)

Table I. Advantages of Supracervical Abdominal Hysterectomy

1. better support for the vaginal vault , so perhaps less risk of patients requiring further surgery in the future for prolapse.
2. shorter operating time.
3. less blood loss—perhaps a major issue , especially when operating on Jehovah's Witnesses, anemic patients, or the elderly. (JR Scott et al in their 1997 article give a rate of hemorrhage of 2% for TAH, with a range of 2% to 15.4%, versus SCH with a rate of 2%, and a range of 0.7% to 4.0%). [4] *
4. less risk of injuring the ureters.
5. less risk of infection, since the vagina is not entered. (JR Scott et al give a rate of infection with TAH to be 3% with a range of 3% to 20%, versus the SCH rate of 1.4%, with a range of 1% to 5%.) [4]
6. possible benefit to patient or partner regarding increased sexual gratification if the cervix is left in place.
7. being able to make the patient happy — by granting her request to keep all of the "original equipment" she can, whether or not it is of any provable benefit to her.

*Note: Talking about the average number of ccs of blood loss in TAHs vs SCHs is in my opinion missing one of the major points re blood loss. Particularly in Jehovah's Witness patients with whom we have contracted to not give blood under any circumstances, the main issue is to avoid major hemorrhage. One of the areas where major hemorrhage is most likely to occur during hysterectomies is in the dissection of the lower cardinal ligament, if a uterine vessel is injured. It is here that the ureter is very close, and the surgeon may be understandably timid in dealing with a bleeding vessel for fear of injuring the ureter —leading rapidly to large bloood losses. Performing a SCH thus completely avoids one of the major areas where hemorrhage occurs, by allowing the uterine vessels to be clamped, cut and ligated, and a small bite of the upper cardinal ligament to be clamped, cut and ligated,both at a location where visibility is good and the ureters are relatively far away, and then completely avoiding those vessels for the remainder of the case.

Table 2 Contraindications to Supracervical Abdominal Hysterectomy

Absolute:

1. cervcial cancer
2. recurrent high grade squamous intraepithelial neoplasia

Relative:

1. uterine prolapse
2. multiple sexual partners (increasing the risk of HPV or HIV exposure)
3. smoking (smoking being a major risk factor for cervical cancer)
4. history of previous cervical SIL or abnormal pap smears
5. non-compliant patient who may not continue to have regular Pap smears.

Elective abdominal supracervical hysterectomy may be commonly performed already in some areas of the country. I believe that it should be — as did Dr. Nichols. I spoke to him about this subject on May 19, 1998.

He kindly sent me a letter on the subject on the same day. It read :

> *Dear Bob:*
>
> *Enclosed herewith please find a copy of some remarks pertaining to subtotal hysterectomy from the 1993 edition of Gynecologic and Obstetric Surgery.[5] Yes, we need more of them, provided only that the surgeon doing them is also capable of doing a safe total abdominal hysterectomy when the occasion requires.*
>
> *Cordially,*
> *David H. Nichols, MD*

Dr. David Nichols died on July 15, 1998. He had been operating that morning, singing in the operating room , and doing what he loved to do.

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Physicians Newly Licensed in Rhode Island During 1998

Nikki Samaras Deary, Milton W. Hamolsky, MD, and Stanley M. Aronson, MD, MPH

At yearly intervals since 1993 the Rhode Island Board of Medical Licensure & Discipline has published a brief demographic profile of those allopathic physicians who were newly licensed to practice medicine in the state of Rhode Island and Providence Plantations during the preceding 12 months. The current summary report to the medical profession of this state, covering the 1998 licensees, is the ninth assembled in behalf of the Board.[1-8]

During 1998, 322 new physicians were granted full licenses by the Board. Physicians renewing their Rhode Island licenses numbered 3,150 resulting in a total of 3,472 licensed Rhode Island physicians. [This number does not include those physicians undergoing residency training in the state who have been issued limited licenses.] The total number of physicians with active licenses [3,472] and the number newly licensed [322] are the largest numbers ever recorded in Rhode Island. The number of newly licensed [now constituting about 9.3% of the total number of physicians in the state] is 5.6% greater than the number who had registered for licensure in 1997.

This significantly increased number of licensed physicians in 1998 may give a false impression that more health coverage for Rhode Islanders will inevitably ensue. Increasingly, newly licensed physicians tend to hold concurrent licenses in more than one state [see below for data]. Some physicians with newly acquired Rhode Island medical licenses, particularly subspecialists, maintain their principal offices in an adjacent state and use their Rhode Island medical license merely to see an occasional in-state patient. In addition, many physicians now apply for medical licenses not primarily to practice medicine but rather to work in some non-clinical administrative position. In addition, many physicians still in residency training within the Rhode Island hospital system seek full licensure which allows them to provide occasional nighttime coverage for other practicing physicians ["moonlight-

Table 1. Physicians Newly Licensed in Rhode Island ,1992 to 1998: Age and Gender

	1992	1993	1994	1995	1996	1997	1998
No. males	130	190	151	180	190	210	225
No. females	69	69	83	84	10	95	95
Total	199	259	234	264	294	305	322
Percent females	*34.7%*	*26.6%*	*35.5%*	*35.5%*	*35.4%*	*31.1%*	*30.4%*
Aver. age, males	36.5	37.3	37.6	37.6	37.1	38.2	39.7
Aver. age, females	34.2	36.3	35.1	34.5	34.7	34.3	35.1
Percent 61 or older							
males	*4.6%*	*1.6%*	*4.0%*	*3.3%*	*4.7%*	*2.4%*	*3.5%*
females	*0%*	*0%*	*1.2%*	*0%*	*0%*	*1.1%*	*0%*
Total no. licensed physicians*	2,835	2,898	3,267	3,286	3,303	3,340	3,472

*: No. physicians [M.D.] with active Rhode Island licenses as per January 2 of the year signified.

Table 2. Physicians Newly Licensed in Rhode Island, 1993 - 1998: Places of Birth

Place of Birth	1993	1994	1995	1996	1997	1998
Connecticut	8	8	12	12	10	17
Massachusetts	27	23	31	37	25	34
Maine	2	0	0	1	0	1
New Hampshire	1	1	1	0	1	0
Rhode Island	23	17	17	18	28	18
Vermont	1	1	0	1	1	2
New England, total	62	50	61	69	65	72
	[23.9%]	[21.4%]	[23.1%]	[23.5%]	[21.3%]	[22.4%]
District of Columbia	5	4	2	3	3	0
Maryland/Delaware	2	4	4	2	3	6
New Jersey	14	14	11	17	14	12
New York	56	45	54	52	60	55
Pennsylvania	12	13	10	18	7	17
Middle Atlantic, total	89	80	80	92	87	90
	[34.4%]	34.2%]	30.3%]	[31.3%]	[28.5%]	[28.0%]
Southern states	16	15	13	10	18	10
North Central states	9	22	12	14	9	27
Midwestern states	13	11	22	21	18	14
Mountain states	5	1	3	4	3	2
Pacific states	5	10	7	7	8	7
Other states, total	48	59	57	56	56	60
	[18.5%]	[25.2%]	[21.6%]	[19.0%]	[18.4%]	[18.5%]
Canada	4	2	6	6	6	8
	[1.5%]	0.9%]	[2.3%]	[2.0%]	[2.0%]	[2.5%]
Countries, other	56	43	60	71	91	92
.	[21.6%]	18.4%]	[22.7%]	[24.1%]	[29.8%]	[28.6%]
Total	259	234	264	294	305	322

Table 3. Physicians Newly Licensed in Rhode Island: Location of Attended Medical Schools

	Year of Rhode Island Licensure							
Region	before 1992	1992	1993	1994	1995	1996	1997	1998
New England	25.6*	29.4	25.9	24.4	25.1	25.5	23.6	27.6
Mid Atlantic	29.3	26.9	31.7	33.3	29.5	29.6	31.5	27.0
USA, other	17.1	26.9	19.3	27.3	26.9	22.4	20.7	21.1
Canada	2.0	1.5	3.9	0.9	3.4	3.1	3.0	2.5
USA+Canada,total	*74.0*	*84.7*	*80.8*	*85.9*	*84.9*	*80.6*	*78.8*	*78.3*
Latin America**	5.4	4.5	5.0	1.7	4.2	4.4	5.2	3.7
Asia	5.8	5.0	3.9	3.0	6.1	6.8	8.5	8.1
Middle East***	3.5	2.5	3.5	3.4	1.7	3.1	2.6	3.4
Africa	0.3	0.4	0.7	0.4	<0.1	0	1.0	0.6
Europe, west	8.9	1.5	4.2	4.7	2.2	3.1	1.3	3.4
Europe, east	2.0	1.5	1.9	0.9	<0.1	2.0	2.6	2.5
Other, total	*25.9*	*15.4*	*19.2*	*14.1*	*14.2*	*19.4*	*21.2*	*21.7*

* : percent **: includes Caribbean island medical schools ***: Includes Egyptian medical schools

ing.”] Therefore, the number of licensed physicians, without knowing the extent to which they each render primary or secondary care, tells us little about the adequacy of medical coverage within the state.

Gender and Age:

Table 1 summarizes the sex distribution and average ages of the newly licensed physicians for each year since 1992.

The yearly number of newly licensed female physicians continues to rise although not as dramatically as the rise in the number of male physicians. As a result, the female fraction in each yearly physician-cadre, has declined from 35.5% in 1994 to 30.4% in 1998.

No discernible trends are evident in the average ages of the newly licensed physician cohort. Women physicians, on average, continue to be younger by a few years. Part of this discrepancy in average age is due to the selective presence of male physicians 61 years of age or older who constitute 3.5% of the male contingent of newly licensed physicians. The oldest woman physician in the 1998 cohort was 58 years of age. Most of the newly licensed older male physicians were recruited to Rhode Island for administrative or academic posts [particularly in geriatric medicine.]

The newly collected data confirm that the character of medical practice is changing. A pathologist, with primary offices in a midwestern state, now holds a Rhode Island license thus allowing him to interpret locally derived surgical biopsies; and a diagnostic radiologist, with a primary office in a southern state, may now view radiographs transmitted electronically from Rhode Island. The Board has no information as to the number of Rhode Island-based physicians who have expanded their practices by establishing satellite offices in other states.

Places of Birth:

Table 2 summarizes the places of birth of the 322 physicians newly licensed in 1998. The numbers born in New England during the past seven years have varied between 21 and 24%. The numbers born in the middle-Atlantic states have diminished in recent years although New York still supplies more doctors to the Rhode Island community of physicians than any other

Table 4. Physicians Newly Licensed in Rhode Island: Most Commonly Attended Medical Schools*

Licensed	Licensed In					
prior to1993	1993	1994	1995	1996	1997	1998
Tufts	Brown	Brown	Brown	Brown	Brown	Brown
Brown	Tufts	SUNY	Tufts	NJersey	SUNY	Boston
Boston	SUNY	Tufts	SUNY	SUNY	Boston	Tufts
SUNY	Boston	Boston	UMass	Boston	UMass	Harvard
Bologna	Columbia	Columbia	Boston	Tufts	G’town+	SUNY
G’town	Penn	Dartmouth	NYMed	Vermont	NJersey	NJersey
Harvard	NJersey	NJersey	NJersey	G’town	Tufts	Penn
NYMed	Dartmouth	Cornell	McGill	UMass	Einstein	UConn
Vermont	Harvard	Vermont	Yale	Jefferson	Penn	Dartmouth
Jefferson	UMass	UMass	Columbia	Einstein	Mt Sinai	Vermont
Columbia	Jefferson	GeoWash	Einstein	UConn	UConn	UMass
Yale	NYMed	NYMed	G’town	Penn	Vermont	Cornell

*: listed in rank order **: represents medical graduates of all four geographically separated medical campuses of State University of New York [Brooklyn, Syracuse, Stony Brook, Buffalo.] ***: Represents medical graduates of both geographically separated medical campuses [Newark and Rutgers.] +: Georgetown

state [and has done so for as long as the Board has been maintaining records.] Massachusetts currently provides 10.6% of the newly licensed cohort; and Rhode Island, 5.6%.

The majority of Rhode Island-born physicians elect to practice in states other than Rhode Island.

The number of newly licensed physicians born in other nations exceeds 28%. The largest single group (33.9% of the foreign-born) were native to Asia, principally India and Pakistan. In the last few years, as a manifestation of the increasing cultural diversity of the Rhode Island community, more of this state's newly licensed physicians were born in the Indian subcontinent than in Rhode Island.

GEOGRAPHIC SITES OF MEDICAL EDUCATION:

The locations of the medical schools attended by the 1998 newly licensed cohort of physicians are summarized in Table 3. There is little change when compared with the distribution noted in 1997. The medical schools of western Europe, principally Italy, and the Cartibbean region no longer provide a significant share of the newly licensed physicians of Rhode Island. Asian medical school graduates, particularly from India, now constitute about 8% of the newly licensed.

Place of birth remains a major determinant in the geographic site of medical school attended. Virtually all Rhode Island-licensed physicians who attended international medical schools were born in the country containing that medical school.

Table 4 lists the medical schools most frequently attended by the newly licensed 1998 cohort. In the early decades of this century, Harvard provided the largest contingent of practitioners for Rhode Island. After World War II Tufts, Boston University, Georgetown, Jefferson and Bologna educated the bulk of this state's new physicians. Since 1993, however, Brown's medical school has consistently provided the plurality of physicians for Rhode Island. Over 14% of the Rhode Island physicians younger than 35 received their formal medical school training at Brown. Each one of the nine medical schools of New England is represented by at least one graduate in the 1998 cohort of physicians. Indeed, graduates of 43 different United States and five Canadian medical schools [McGill, Toronto, Western Ontario, Montreal, Queens] are also represented in this group of 322 physicians. The state-funded medical schools in New York, New Jersey, Connecticut and Massachusetts, collectively, continue to educate about 15% of Rhode Island's newer physicians. Graduates of the Ivy League medical schools, with the exception of Brown, Harvard and Pennsylvania, had not chosen to practice in Rhode Island as readily as in the past; but this, too, seems to be changing with only Columbia and Yale not represented within the top twelve medical schools in the current cohort.

LICENSURE PATHWAYS AND NUMBERS OF ACTIVE MEDICAL LICENSES:

The tests of the National Board of Medical Examiners [and its successor tests, the United States Medical Licensure Examination] provided the needed certification for over 80% of the newly licensed physicians. The remainder presented certification from the state-administered FLEX examinations.

Applicants for medical licensure in Rhode Island are asked whether they hold currently active medical licenses in other states or jurisdictions. Of the male physicians who obtained their MD degree within the past six years, 37.1% held one or more out-of-state licenses; and of those who obtained their MD degrees more than six years ago, 96.0% held one or more out-of-state licenses. Comparable figures for female physicians were 42.1% for those who obtained the MD degree within six years, and 93.7% for those who graduated from medical school more than five years ago. Thus, the 225 newly licensed male physicians collectively held 356 out-of-state medical licenses [1.58 licenses per physician] and the 95 newly licensed female physicians collectively held 132 out-of-state licenses [1.39 licenses per physician.] Seven years ago, the average number of licenses held by male physicians far exceeded those held by their female counterparts, partly, it was presumed, because women physicians were allegedly less mobile than their male colleagues. In the last hemi-decade, however, these gender-oriented rates have progressively converged to a point where there is now an inappreciable difference in rates between genders.

The newly licensed physicians holding the largest numbers of out-of-state licenses tend to be employed by companies filling *locum tenens* positions. One such physician holds licenses in 33 different states. Yet another physician with large numbers of licenses supervises a chain of renal dialysis centers operating in many states.

POSTGRADUATE SUPERVISED HOSPITAL TRAINING:

Physicians applying for medical licensure in Rhode Island are required to provide the Board with the specialty, duration and location of all of their supervised postgraduate experiences [internship, residencies, fellowships, research positions]. Since many of the candidates for licensure are still undergoing residency or fellowship training, the average lengths of formal postgraduate training for this cohort was determined by considering only those

Table 5. Physicians Newly Licensed in Rhode Island: Distribution in Percent by Gender, Specialty and Year of Licensure [1993 - 1998]

Specialty	Males						Females					
	1993`	1994	1995	1996	1997	1998	1993	1994	1995	1996	1997	1998
Primary care*	29.5	40.4	28.3	39.4	32.3	24.4	58.0	63.9	47.6	43.3	63.8	49.5
Non-surgical specialties**	26.8	21.9	36.7	21.1	25.8	33.3	14.5	14.5	30.0	32.7	20.2	25.8
Surgical specialties***	21.6	13.2	15.6	20.0	16.4	20.0	10.1	4.8	6.0	11.5	4.3	10.3
Emergency medicine	5.3	5.3	4.4	4.2	10.5	4.0	4.4	3.6	0	0	5.3	3.1
Institution-based specialties+	16.8	19.2	15.0	16.3	15.0	18.3	13.0	13.3	16.5	12.5	6.4	11.3

*: includes family medicine, general practice, pediatrics, obstetrics/gynecology, and internal medicine without a defined subspecialty. **: includes internal medicine subspecialties, psychiatry, neurology and dermatology. ***: includes general surgery, orthopedics, ophthalmology, otolaryngology, urology, neurosurgery and plastic surgery, +: includes pathology, radiology, physical medicine, anesthesiology and administrative medicine.

newly licensed physicians with a minimum of six years following the awarding of their MD degrees. Male physicians completed, on average, 4.0 years of supervised, accredited residency training, and female physicians, 3.9 years.

Again, considering solely those who have have been through six or more years of post-MD experience, fellowship training, typically in a subspecialty, was undertaken by 33.6% of male physicians [with an average of 2.1 years of fellowship] and 42.4% of female physicians [with an average of 1.6 years of fellowship.]

Table 6. Newly Licensed Physicians, 1998: Most Frequently Selected Residency Training Sites

Rhode Island
- Rhode Island Hospital
- Brown University, combined programs
- Hasbro Hospital
- Memorial Hospital
- Butler Hospital
- Miriam Hospital
- Roger Williams Hospital
- Women & Infants Hospital

Massachusetts
- Harvard, combined programs
- Mass General Hosp, Brigham & Women's Hosp
- Boston University Medical Center
- University of Massachusetts Hospitals
- Beth Israel-Deaconess Hospital
- St. Elizabeth Hospital

New York
- State Univ NY/ Kings County Hospital
- Montefiore Hospital
- Mt Sinai Medical Center

Connecticut
- Yale-New Haven Hospital
- Hartford Hospital

Maryland/DC

- Johns Hopkins Hospital
- Walter Reed Medical Center
- Naval Medical Center, Bethesda

SPECIALTY DISTRIBUTION:

Table 5 summarizes the specialties and subspecialties declared by these 322 newly licensed physicians comparing these data with the preceding five cohorts of newly licensed physicians. Substantial variations appear from year to year, making it difficult to detect any trends. Certainly, the percent of women physicians selecting primary care medicine has remained consistently greater than the percent of males entering primary care. The percentages of either male or female physicians entering the non-surgical specialties tend to be about the same; but males continue to dominate the surgical specialties.

Table 6 lists the institutions most frequently selected by the newly licensed physicians for their residency training. Rhode Island Hospital and the Brown University multi-hospital programs continue to dominate the roster. Military installations (e.g., Walter Reed Medical Center and the Naval Medical Center in Bethesda) provided residency programs for nine male and two female physicians. These military training sites are located in the District of Columbia, Mary-

Table 7. Physicians Newly Licensed in Rhode Island: Geographic Location of Residencies/Fellowships

Location	Residency Training		Fellowship Training	
	1997	1998	1997	1998
Rhode Island	30.2%	27.8%	17.1%	14.3%
Massachusetts	13.8	18.3	34.1	41.3
Connecticut	7.5	4.9	3.7	5.4
New York	15.4	10.7	10.0	7.6
New Jersey	2.6	0.7	0	1.1
Pennsylvania	6.2	5.3	8.5	4.3
California	2.6	3.2	1.2	4.3
Other	17.1	23.1	20.5	17.4

Table 8. Physicians Newly Licensed in Rhode Island: Board Certification

Specialty	Percent Board Certified
Family medicine	63.6
Pediatrics	42.4
Internal medicine	47.4
Obstetrics/gynecology	38.9
Primary care medicine*	47.7
Non-surgical specialties*	60.5
Surgical specialties*	58.1
Emergency medicine	53.6
Institutional specialties*	59.0
Total	54.1

*: See footnotes in table 5 for definitions.

land, Hawaii, California, Colorado, Texas and New Hampshire.

Table 7 summarizes the geographic locations of the residency and fellowship programs for both the 1997 and 1998 cohorts of newly licensed physicians. In contrast to the wide geographic distribution of places of birth [table 2] and sites of formal medical education [table 3], the residency/fellowship programs tend to be based in or near Providence. In 1997, 51.5% of residency sites were based in Rhode Island or the two contiguous states of Massachusetts and Connecticut. In 1998, this rate was virtually the same [51.0%]. The fellowship sites showed a similar clustering around the three southern New England states [54.9% in 1997, and 61.0% in 1998.] In recent years all of the fellowships had been based in the United States. In the 1998 cohort, one candidate completed an accredited fellowship in England and another in Canada.

SPECIALTY BOARD CERTIFICATION:

Many of the newly-licensed physicians were still in residency/fellowship training at the time of licensure application and hence were not eligible to sit for the certifying specialty examinations. For those male physicians with fewer than six years of experience since medical school graduation, 9.9% were Board-certified; amongst female physicians with fewer than six years of postgraduate experience, 23.0% were Board-certified. Amongst those with six or more years of experience since graduation, 70.6% of male physicians were Board-certified and 70.7% of female physicians were Board-certified. When these data are compared with figures from the prior six years, two trends are apparent: First, in the past, the rate of Board-certification had always been higher amongst male physicians. This is no longer the case. Second, Board-certification had always been substantially higher amongst physicians educated in the United States/Canadian medical schools. The Board-certification rate for graduates of international medical schools, while still lower, is rapidly approaching the rates achieved by the United States/ Canadian licensees.

COMMENT:

This is the ninth in a series of summary papers designed to provide the established physicians of Rhode Island with a demographic profile of their newly licensed colleagues. No attempt is made to employ these data to predict the future of medicine in Rhode Island. Given the multitude of forces currently manipulating the medical profession, it is foolhardy to think that an understanding of the profession's future lies buried in these statistics. Five conclusions, though, may be safely expressed: First, Rhode Island continues to attract significant numbers of well-educated, well-trained physicians. Second, gender differences, so evident in years past, are rapidly disappearing. Third, the majority of these newly licensed physicians come to Rhode Island, not as independent practitioners but as salaried professionals beholden to a hospital, an HMO or a medical school. Fourth, the academic hospitals of this community, particularly Rhode Island Hospital, can note with pride their dominant role in providing the bulk of supervised graduate education for the practitioners of this state. And fifth, the medical school at Brown has fulfilled its promise, made 25 years ago, that it would become, within a few decades, the major source of practicing physicians for Rhode Island.

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THE CREATIVE CLINICIAN: CASE OF THE MONTH

"The practice of medicine is an art, not a trade; a calling, not a business . . ." – WILLIAM OSLER, Aequanimitas

Editor: Anthony Mega, MD; Co-editor: Fred J. Schiffman, MD

Hodgkin's Disease in Patients with HIV/AIDS: Case Report and Literature Review

Fadi F. Mansourati, MD, and Valerie E. Stone, MD, MPH

Human immunodeficiency virus (HIV) infection, like other immune deficiency states, is associated with certain malignancies, like non-Hodgkin lymphoma (NHL), Kaposi's sarcoma, and invasive cervical cancer.[8] The incidence of each of these conditions is clearly higher in the HIV-infected population. The association between Hodgkin's disease (HD) and HIV remains debatable. While some studies found an increased incidence of HD with HIV[4,5], others did not. The peak age for both entities, being so similar, makes establishing a relationship more difficult. Many studies, however, have consistently found that in HIV-infected individuals, HD tends to present with unfavorable histology and at a more advanced stage. It also tends to occur in later stages of HIV infection, in those with lower CD4 counts and a history of OIs. Some authors have proposed including HD in the AIDS case definition.[9,10] We present a case of HD in a patient known to have AIDS, and summarize the literature on HD and HIV/AIDS.

Abbreviations Used:

ABVD	adriamycin, bleomycin, vinblastine, doxorubicin
AZT	zidovudine
CT	computed tomography
HD	Hodgkin's Disease
HIV	human immunodeficiency virus
NHL	non-Hodgkin lymphoma
MOPP	mechlorethamine, vincristine, procarbazine, prednisone
OI	opportunistic infections

CASE PRESENTATION:

A 64 year-old Hispanic man, seropositive for HIV since 1994, presented with a two-week history of protracted vomiting, abdominal pain, generalized weakness, night sweats, and 15 lb weight loss over three months. He acquired HIV via heterosexual contact, with nadir CD4 count of 12 cells/ml and most recent count of 174 cells/ml. His other significant medical problems included peripheral vascular disease and hypertension. His medications included hydrochlorothiazide, ASA, zidovudine (AZT), zalcitabine (ddC), indinavir, clarithromycin, and dapsone. His vital signs were normal on admission and physical exam revealed a 1.5x2 cm right axillary soft, non-tender, mobile lymph node. Laboratory studies revealed pancytopenia, elevated LDH with otherwise normal liver enzymes. There was no growth on blood culture bottles with regular and special (fungal and acid fast bacilli) media. A chest CT scan showed multiple small mediastinal lymph nodes, and one larger (1.9cm) node in the aortopulmonic window. An abdominal CT showed scattered small retroperitoneal lymph nodes. A bone marrow biopsy showed hypocellularity, with no specific changes otherwise. The patient was discharged home after changing his antiretroviral regimen (which included discontinuing AZT).

Two weeks later, the patient was re-admitted for high grade fever, vomiting, and worsening of his previous symptoms. His temperature fluctuated from normal to as high as 105.2°F. An extensive work-up and repeated cultures failed to detect an infectious etiology. A repeat abdominal CT revealed new splenomegaly with subtle small foci of decreased attenuation.

The right axillary lymph node was excised, and the pathology was compatible with Hodgkin's disease of mixed cellularity subtype. A review of further sections of the earlier bone marrow specimen also revealed Reed-Sternberg cells. The patient was started on adriamycin, bleomycin, vinblastine, doxorubicin (ABVD) at a reduced dose. The patient has now undergone four courses of this regimen and is doing well; his course has been complicated by several episodes of C. difficile colitis.

DISCUSSION

Even though HD is not an AIDS-defining condition, multiple studies have suggested a higher incidence of HD in patients infected with HIV. Definitive data to support this association are lacking, though. The clinical importance of the above is that in the appropriate settings when a lymphatic malignancy is suspected, NHL should be high on the differential diagnosis in HIV-infected patients; HD, however, should also be a consideration. Of the studies that looked at HD in HIV-infected populations, the two largest series of cases were reported from Italy in 1995[1] and Spain in 1993.[3] Both suggested a strong association of HD to HIV-infected injection drug users (79% of cases in the Italian series). The reason for this association is unknown. One hypothesis is that a blood-borne pathogen may play a causative role in HD.[2,3] Median age was 29 years compared to 34 years in non-HIV-infected

HD patients, with men comprising 90% as compared to 56% in non-HIV-infected persons[1], which probably reflects gender distribution in HIV. It is also important to realize that HD in this particular group of patients has distinguishing features, including a more aggressive histology, more advanced stage at time of presentation with a higher incidence of extranodal disease, and a higher incidence of B symptoms. HD tends to be more aggressive in HIV-infected patients than the general population, and advanced clinical stages at time of presentation are rather the rule. Stage IV accounted for 52% of patients at presentation in the Italian group for example;[1] and on average, 74-94% of all patients presented with either stage III or IV[1,3,7,10,17,19-25] - significantly higher than HD in the general population in which approximately 40% present with stage III or IV.[26] Another aspect of HD which differs in HIV-infected persons is that B symptoms are significantly more common: 70-100% of HIV-infected patients with HD present with B symptoms,[7,14,22-24] compared to 35% in those without HIV.[27] Higher incidence of bone marrow involvement at the time of presentation is also another characteristic of HD in HIV, with such involvement recorded in 41-77% of cases[1,3,14,7,22,23] compared to 35% in historic controls.[26] Also, only 33% of HD patients infected with HIV had evidence of mediastinal involvement, as compared to 58% in non-HIV.[1] Histopathologically, the main subtypes (nodular sclerosis and mixed cellularity) seem to occur in reversed frequency in HIV-infected cases.[1] The mixed cellularity (MC) type was clearly the most common (30-100%), followed by nodular sclerosis (NS) which was found in 0-54%;[1,3,7,14,15,22-24,28] this contrasts with the historic control incidence in non-HIV patients (NS 52-62% and MC 23-25%).[27] Lymphocyte depletion type was more common in association with HIV (21% and 21.7% in the Italian and the Spanish groups, respectively),[1,3] as compared to 3-6% in HD in the historic controls.[27] One possible explanation is that MC and LD (more common in HIV-infected than in non-HIV) have stronger association with EBV than the other subtypes[1,6], which also seems to be more common in HIV-infected HD patients than the overall HD population. It is not clear, however, whether the higher incidence of EBV in HIV-infected individuals is a cause or result of the increased frequency of MC and LD subtypes. All of these atypical findings suggest that HD in HIV-infected patients is different from classic HD (in non-HIV). The above findings seem so characteristic of HD in HIV-infected individuals, that some authors have suggested considering HD of mixed cellularity or lymphocyte depleted subtypes an AIDS-related malignancy when it occurs in HIV-infected individuals with atypical clinical patterns of presentation and advanced stage disease.[1]

in HIV-infected patients with a constellation of symptoms that include fever, weight loss, and lymphadenopathy, HD should be considered along with the more commonly encountered pathologies

Median survival in the two largest series of HIV/AIDS patients with HD was 15 months from diagnosis of HD[1,3]; similar survival rates were found in other series.[7,14,24,30] The survival was significantly higher for those with CD4 counts higher than 250 cells/ml, in patients without AIDS at time of diagnosis of HD, and in those who achieved complete response following chemotherapy.[1,2,7] Presence of B symptoms, anemia, neutropenia, lymphopenia, lower CD4 counts, and high serum alkaline phosphatase levels on the other hand, were poor prognostic signs.[3]

In non-HIV patients with HD, response to standard therapeutic regimens such as MOPP (mechlorethamine, vincristine, procarbazine, prednisone) or ABVD (adriamycin, bleomycin, vinblastine, dacarbazine) is 89% and 8-year disease-free survival is 73% [29]. In HIV- infected HD patients, on the other hand, the complete remission rate is only 48-64%.[3,7,14,22,30] Major myelotoxicity from chemotherapy is more frequent with HIV,[31] and opportunistic infections were a major cause of morbidity and mortality in HD patients with HIV.[1,7,22-24]

In summary, even though it is not clear whether HD is more common in HIV-infected patients, HD in these individuals seems to have characteristic features that distinguish it from that in non-HIV infected patients. These features include more aggressive disease, less favorable histologic types, stronger association with EBV, and a poorer prognosis. Based on this review, we believe that in HIV-infected patients with a constellation of symptoms that include fever, weight loss, and lymphadenopathy, HD should be considered along with the more commonly encountered pathologies such as atypical mycobacterial infection, non-Hodgkin lymphoma, histoplasmosis, and extrapulmonary tuberculosis. The possibility of HD should also be entertained in HIV-infected patients with symptoms and signs of specific organ involvement (especially bone marrow failure). Further data is needed to examine any changes in the natural history of HD in HIV-infected patients who are receiving effective antiretroviral therapy.

References available upon request

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Letter to the Editor:

I have been in practice since 1972. The following incidents add up to a frightening lesson.

Three times my children needed specialty care. In the first instance, the specialist's office staff told us that a certain fee was due at the time of the visit. When the visit was over, we were charged more than the quoted amount. The staff told us the fee was reduced for professional courtesy. That's some courtesy.

One surgeon performed an operation on one of our sons. After the operation, the surgeon instructed us to bring our son to the office for a checkup the following week. Another member of the group did the follow-up examination. He had the front desk charge us a fee.

In another case, the surgeon charged us for two follow-up visits. In both cases, the surgeons participated in our insurance plan and were supposed to accept the global fee for management of the entire case. The surgeons and the office staffs had to know it. This is just base greed. No wonder the public is upset.

I have also worked in a hospital in the area, where the duties of the night physician include performing histories and physicals on admitted patients. The hospital in turn bills the insurance carrier for the service based upon the H&P. The level of billing, of course, depends upon the extensivesness of the H&P. I know that the H&P as documented are not being performed. Due to the nature of the cases it is highly unlikely an audit would ever discover it.

These are just the abuses encountered by one family. Add to that the number of times physicians put in an unjustified procedure or diagnostic code so that "they'll pay for it" or a procedure is performed because "they pay for it" and you realize how far we have deteriorated.

At every medical meeting there will be moaning, groaning and gnashing of teeth over what the third parties have been doing while the complainers turn around and accept their money. They can't have it both ways. After my experience, I don't want to hear any more complaining. From what I can see, we're getting what we deserve.

– Roderick. T. Beaman, DO
Cranston, RI

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Rhode Island
Quality Partners, Inc.

Update on the SCRIPT Project

Edward Westrick, MD, MS

The SCRIPT Project is a national effort of the Coalition for Quality in Medication Use. SCRIPT is an acronym that stands for Study of Clinically Relevant Indicators of Pharmacologic Therapy. The project is funded by the Health Care Financing Administration (HCFA) and is staffed by the Joint Commission on the Accreditation of Health Care Organizations (JCAHO). The Coalition is made up of more than 50 national organizations including government and private sector partners. The AMA, PROs, HCFA, JCAHO, NCQA, AAHP, APhA and AHCPR steer the project activities on a week to week basis. Specialty societies have been involved in the coalition's expert panel and committees.

The purpose of the project is to create a common set of medication use quality indicators for adoption by health care organizations across the country. The Coalition met for the second time just last month to review the progress of the project and to plan future activities. In this month's column I review this progress and outline next steps.

The project has developed a methodology for identifying evidence-based and consensus-driven performance measures related to medication use in the ambulatory setting. This methodolgy includes the establishment of criteria for evaluating performance measures and the convening of a technical expert panel to review measures according to these criteria. An applications group is making recommendations for the feasibility of measurement. Two new groups were formed at the last coalition meeting. One will focus on making recommendations for the use of performance measures for potential use in accountability and for quality improvement. Another group will focus on the measurement model.

The criteria for evaluating measures have been reported in an earlier edition of this column[1] and are reproduced here. Performance measures should focus on clinical topics that are important and meaningful. There should be a relatively high disease burden (prevalence and severity), and potential for improvement. Measures should be based upon guidelines supported by scientific evidence and by expert consensus when sufficient scientific evidence is lacking. Measure components should be well-specified including procedures for data collection and analysis. Measures should be tested with scientific rigor to establish reliablity and validity. Measures should focus on clinical topics where there are opportunities to provide cost-effective care. The measurement process must be feasible, reducing data collection burden as much as possible. The measures should be interpretable by consumers, clinicians, and researchers.

Abbreviations Used:

AAHP	American Association of Health Plans
AHCPR	Agency for Health Care Policy and Research
AMA	American Medical Association
AMAP	American Medical Accreditation Program
APhA	American Pharmaceutical Association
HCFA	Health Care Financing Administration
HEDIS	Health Plan Employer Data and Information Set
JCAHO	Joint Commission on the Accreditation of Healthcare Organizations
NCQA	National Committee for Quality Assurance
PRO	Peer Review Organization
SCRIPT	Study of Clinically Relevant Indicators of Pharmacologic Therapy

Performance measures can be used for accountability and quality improvement purposes. Performance measurement for accountability can include public reporting and can be linked to financial incentives. Performance measurement for quality improvement is primarily used in providing feedback to monitor performance change over time. The consequences of measurement for accountability can have greater significance. Therefore, the measurements must meet higher standards than those used for quality improvement purposes. SCRIPT will study these issues and make recommendations on how the measures should be used.

The Coalition has decided to start with medication use issues in cardiovascular disease and diabetes mellitus. The clinical conditions include myocardial infarction, coronary artery disease, hypertension, dyslipidemia, congestive heart failure, atrial fibrillation, and diabetes mellitus. The medication use issues to be measured include prescribing, therapeutic monitoring, and compliance. Suggested measures to date include: beta blocker and aspirin use in coronary artery disease, lipid lowering therapy, diuretic and beta blocker use in incident uncomplicated hypertension, ACE inhibitor therapy in CHF and diabetic nephropathy, anticoagulation in atrial fibrillation, and hypoglycemic therapy in diabetes mellitus.

There are currently two approaches under consideration for the measurement model. One approach treats each clinical condition separately with multiple potential indicators for some of the conditions. The second approach groups conditions in clinically meaningful clusters where a score can be derived for groups of patients who are at high risk. This approach might focus on patients with known cardiovascular disease or diabetes mellitus, looking at pharmacologic interventions designed to modify risk. Another cluster worthy of consideration are the patients with CHF who are at high risk for hospitalization due to their disease and due to potential adverse effects of medications. Therapeutic monitoring turns out to be most critical in these patients. The measurement model will be chosen after alpha and beta testing.

The specifications of the measures are currently being developed. Pilot testing should occur later this year, followed by beta testing in 2000. The goal is to release a compendium of core measures at the conclusion of this 2-year project. It is anticipated that this compendium will be updated periodically with quality measures in other clinical conditions and as measurement methodology improves.

This project is complementary to other national activities in the government and private sector related to health care quality improvement: the American Medical Accreditation Program (AMAP), the JCAHO's ORYX iniative, the NCQA's HEDIS, HCFA's Health Care Quality Improvement Program (HCQIP) and the Forum on Health Care Quality Measurement and Reporting. In Rhode Island this project is complementary to our Health Care Accessibility and Quality Assurance Act and the Healthcare Quality Performance Measurement and Reporting Program. These are products of our state legislature and are viewed by many as model legislation for the rest of the country to follow.

Performance measurement in health care will have increasing impact. It is important that this measurement be scientifically sound and not overly burdensome to the system. By standardizing the process and measurement methodology it is hoped that SCRIPT will be able to accomplish these objectives in the area of medication use and to serve as a learning experience and model for efforts to standardize measurement in other health care areas.

Edward Westrick, MD, MS, is the Principal Clinical Coordinator of Rhode Island Quality Partners. He is a member of the clinical faculty of Brown University School of Medicine and the Active Medical Staff of Roger Williams Medical Center. He is currently a PhD candidate at the University of Rhode Island studying pharmacoepidemiology and pharmacoeconomics.

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The author assumes full responsibility for the accuracy and completeness of the ideas presented. This article is a direct result of the Health Care Quality Improvement Program initiated by the Health Care Financing Administration, which has encouraged identification of quality improvement projects derived from analysis of patterns of care, and therefore required no special funding on the part of this Contractor. Ideas and contributions to the author concerning experience in engaging with issues presented are welcomed.

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Health by Numbers

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Jay S. Buechner, PhD

Lyme Disease in Rhode Island, 1990-1998

Edward F. Donnelly, RN, MPH

Lyme disease was identified and named in 1975 and has become the most commonly reported vector-borne disease in the United States. Lyme, Connecticut, where Lyme disease was discovered, is only thirty miles from Rhode Island and is similar ecologically to southern Rhode Island. Lyme disease is caused by the spirochete *Borrelia burgdorferi*, which was identified in 1982 and is transmitted by the bite of the deer tick, *Ixodes scapularis*. The white-footed mouse, *Peromyscus leucopus*, the most common rodent in Rhode Island, is the reservoir for Lyme disease. Most cases of Lyme disease are transmitted by the nymphal form of the deer tick which is active in late May through July. The current surveillance case definition was adopted in 1990 and includes clinical cases that demonstrate *erythema migrans*, the pathognomonic rash of Lyme disease, and cases with positive serology (ELISA and Estern Blot) in the presence of any of a variety of rheumatologic, neurologic, or cardiologic signs and symptoms.

Lyme disease was made reportable in Rhode Island in 1982 and has become one of the most commonly reported infectious diseases in the State. The number of cases has shown an increasing trend with considerable year-to-year variation (Figure 1). Much of this variability is due to variability in the severity of winter weather, the amount of spring rainfall and other climatic conditions, since the vector ticks are quite sensitive to drying and require high ambient humidity to function. A new high of 789 resident cases was reported in Rhode Island in 1998, a seven-fold increase in reported cases since the beginning of the decade. Approximately the same number of cases were reported in the first six months of 1999 as in the corresponding period of 1998 but the record level is not likely to be matched this year because of drought conditions that reduced tick activity by the beginning of July.

Deer ticks can be found in wooded areas throughout Rhode Island. The majority of cases each year have been reported in residents of Washington County, where vector surveillance has confirmed that the greatest risk of exposure exists. Annual tick surveys have also shown year-to-year variability in the distribution of tick densities with a trend of expansion of the area of greatest tick abundance northward, into Kent and Providence counties. There has been a concomitant increase in reported cases from those two counties in recent years.

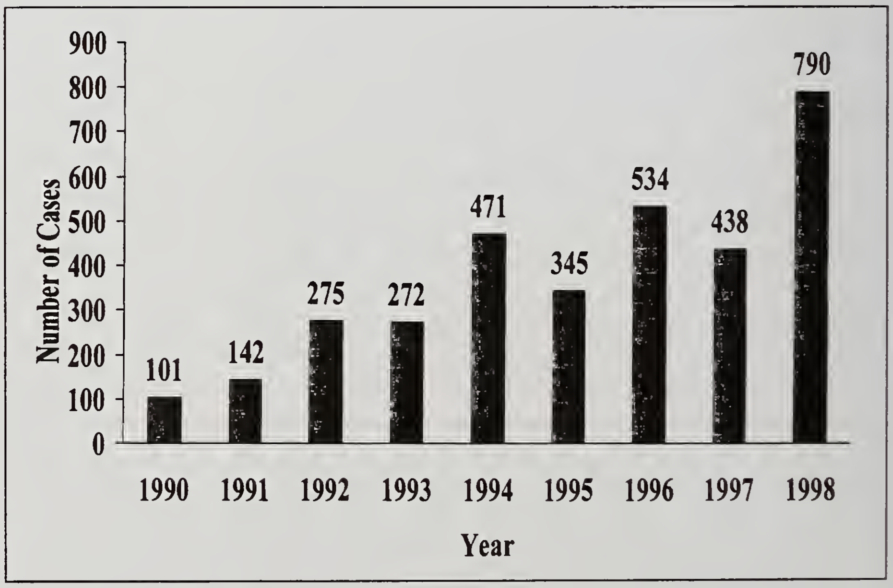


Figure 1. Reported cases of Lyme disease by year, Rhode Island 1990-1998

Reported cases of Lyme disease in Rhode Island are distributed bimodally by age group in a pattern that is consistent from year to year. Few children under five are reported cases while children age five to nine were the largest number of reported cases for any five-year age group in 1998. The number of cases declines sharply among persons in their late teens and twenties and gradually increases to a second high among persons in their forties and fifties. While this distribution reflects the underlying differences in disease occurrence by age group, it may also be influenced by differences in health-care seeking behavior and reporting behavior.

Prevention of Lyme disease is best accomplished through adopting personal protective measures such as avoiding tick habitats, the use of light colored protective clothing (hat, long sleeves, and long pants tucked in socks), the application of insect repellents containing DEET, and a careful inspection for ticks at the end of each day of exposure.

Table 1. Reported Lyme disease rates per 100,000 population by county, Rhode Island 1990-1998.

	Bristol	Kent	Newport	Providence	Washington	Rhode Island
1990	4.1	6.2	11.4	2.2	57.3	10.1
1991	8.2	7.4	15.0	2.5	80.8	14.1
1992	10.2	24.1	16.2	4.1	159.9	27.5
1993	6.1	14.8	18.8	4.9	176.1	27.3
1994	12.3	20.3	28.6	8.2	322.1	47.4
1995	16.4	18.5	21.8	7.2	210.9	34.9
1996	8.2	45.8	32.6	9.9	315.7	54.0
1997	8.2	45.2	25.3	13.7	218.0	44.4
1998	8.1	77.3	39.8	16.7	440.1	79.9

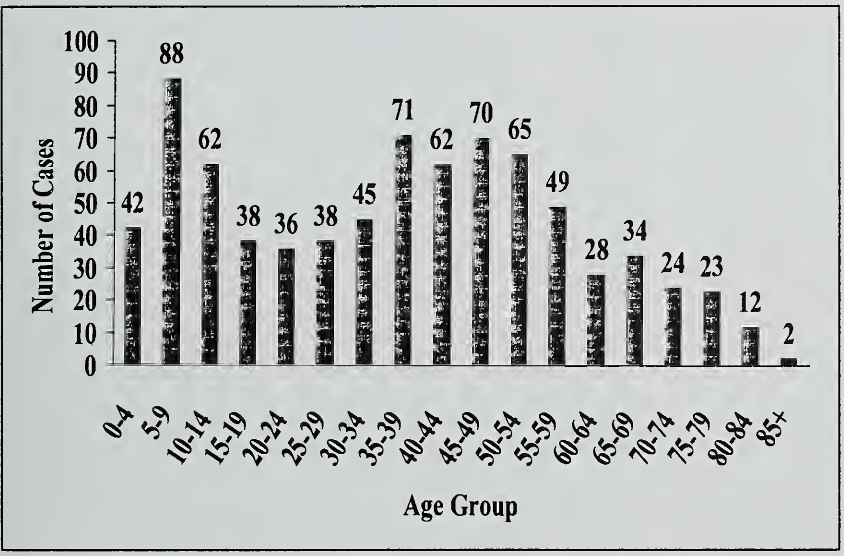
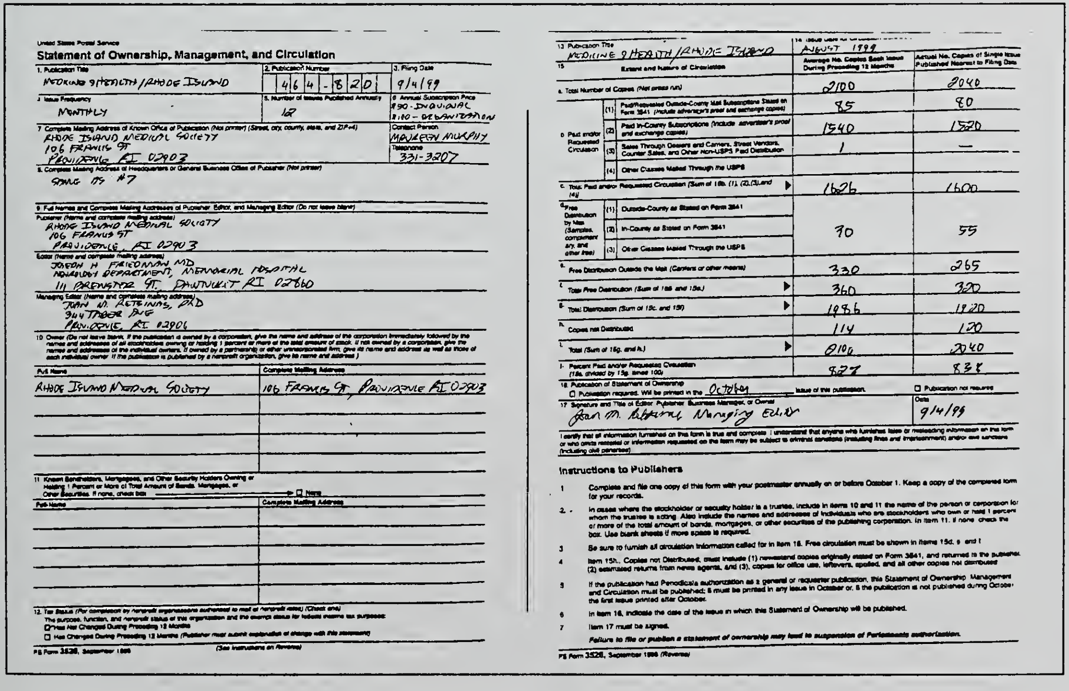


Figure 2. Reported cases of Lyme disease by five-year age groups, Rhode Island 1998.

The Food and Drug Administration has approved a vaccine for use in persons ages 15 to 65 who live in high-risk areas. Currently, only subjects in the vaccine trials have been fully immunized, and the three-dose schedule for the vaccine means that any impact of the vaccine on the incidence of Lyme disease in Rhode Island will only begin to be seen in summer 2000.

Other approaches to disease control include the application of various acaricides on white-tailed deer, who are the primary source of blood meals for adult deer ticks. The deer is attracted to a baited apparatus that automatically applies the agent to its head and neck where ticks attach most densely. These efforts are being piloted by researchers at several locations in Rhode Island and in other states and, if proven effective, will have the added benefit of reducing the abundance of the vector of other pathogens such as those that cause babesiosa and human granulocytic ehrlichiosis.

Edward F. Donnelly, RN, MPH, is a Senior Public Health Epidemiologist, Office of Communicable Diseases.



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PUBLIC HEALTH BRIEFING

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

TB: Prevention and Control Issues, 1999

Edward F. Donnelly, RN, MPH, Thomas Bertrand, MPH, Utpala Bandy, MD, MPH, E. Jane Carter, MD

Early Success: 1900-1984

A summary of public health achievements in the 20th century revealed that the average life expectancy of Americans increased from 42 years in 1900 to 72 years in 1995, fueled, in part, by a dramatic decline in the prevalence of tuberculosis (TB). In 1900, TB caused 10% of all deaths in the United States. By 1984, only 22,255 cases were reported in the country. Improved housing and nutrition early in the century caused much of this decline, but effective antibiotics, introduced at mid-century, also helped.

A Warning: 1984-1991

The decline in the incidence of TB in the United States was interrupted in the 1980s. From 1984 to 1991, reported cases increased by 18% nationwide. Although this resurgence was originally ascribed to the HIV epidemic, the immigration of groups with high levels of latent TB, and transmission within congregate environments such as prisons and hospitals, it was subsequently recognized that the true cause was the systematic dismemberment of the state and local TB control programs which occurred throughout the 1980s. The U-shared curve of concern in public health again held true: as the incidence of TB declined, public concern with support of the programs to control TB declined. Cases were diagnosed late, allowing for ongoing transmission. Cases were not treated to cure, allowing for relapse and continued transmission.

As a consequence of poor control, multi-drug resistant tuberculosis (MDR TB) emerged, further complicating TB control. Inadequate treatment of TB selects for drug resistance. When TB becomes resistant to isoniazid and rifampin, the most effective of the first line agents, cure rates despite appropriate therapy may fall to as low as 60%. Prolonged contagion in these difficult-to-treat patients again allows for transmission of the resistant organisms, breeding the next generation of cases. As antimicrobrial agents have been lost, surgery as an adjunct to improve cure rates for TB has reappeared.

The Rhode Island experience paralleled the national experience. By the mid 1980s, the TB program both in terms of public health department staff support and clinical services at the TB clinic was scaled back. TB cases rose.

Outbreaks of MDR TB in congregate settings such as prisons and hospitals with subsequent transmission to health care workers and the community spurred the rebuilding of TB control programs in the early 1990s.

Abbreviations Used:

CDC	Centers for Disease Control and Prevention
DOH	Department of Health
DOT	directly observed therapy
MDR-TB	multi-drug resistant tuberculosis
TB	tuberculosis
WHO	World Health Organization

Systematic Control: 1992+

Because spontaneous development of multi-drug resistance is a rare event, the keys to prevention of MDR TB are good surveillance, assured completion of therapy with an array of drugs for an appropriate length of time, and strict infection control guidelines to prevent outbreaks in congregate settings. Endorsing this approach, the CDC increased funding to the states for TB prevention and control in 1992, and issued elaborate infection control guidelines for health care facilities in 1994.

Rhode Island used CDC funding to strengthen the TB program in a variety of ways. At the DOH, a TB program manager, an epidemiologist, and an outreach coordinator were employed to assist the 1 existing public health nurse. Mycobacterial testing capabilities at the DOH laboratory were improved to supply state-of-the-art services. Clinical capabilities of the TB Clinic were expanded. Legislative and regulatory changes tightened surveillance for new cases, to ensure that all TB laboratory services utilized current, reliable methods, and to improve timely reporting to the Rhode Island DOH.

In 1992 Rhode Island became only the second state in the nation to develop a universal directly observed therapy (DOT) program following WHO and CDC recommendations. Bilingual outreach workers supply care in this program. Regimens to treat TB are complex. Current CDC recommendations call for therapy to begin with a minimum of 4 drugs (an average daily pill count of 10-13 pills per day!). When patients take TB drugs in inappropriate combinations or miss doses, MDR TB can result. Even the most well-intentioned and well-educated patient can become confused with this complex regimen. In a DOT program each dose of drug is delivered to the patient with patient monitoring for adherence and side effects. DOT not only acts as an effective strategy to assure completion of therapy but also supplies the patient with best care to include earlier recognition of side effects, allowing for earlier regimen adjustments, and to guarantee cure with the minimal doses of medication possible. In addition, the DOT outreach workers improve communication between patients and health care providers: DOT workers seeing patients in their homes recognize other barriers to care which may not be obvious to providers seeing patients in the office. Utilization of DOT has been shown to have multiple benefits in TB control: decreased numbers of relapse, decreased transmission due to rapid and guaranteed sterilization of respiratory secretions, decreased levels of both primary and secondary drug resistance in the communities where DOT is utilized, and finally a cost savings by reducing testing to assess adherence, reducing relapse with retreatment costs, and reducing transmission (which in turn reduces need for contact tracing and evaluation).

Because of this renewed dedication to the long recognized tenets of TB care (early case recogntion, improved diagnostics, and treatment to cure), TB incidence has again declined in the United States. In 1997, the last year for which a final tally is available, 19,861 cases were reported to the CDC, below the number reported in 1984. In Rhode Island, which has a high proportion of immigrants from countries in which TB is endemic, the numbers have remained stable (approximately 67 cases last year, not including 5-6 transfers from other jurisdictions and 1 relapse). New immigrants who have latent TB are at increased risk of reactivation in the first five years after immigration, presumably from the stress of this major life change. Here in Rhode Island, the proportion of cases that are foreign-born continues to be higher than that found nationally. In addition to foreign-born cases, secondary cases are found among young children who are born into immigrant communities. Some children become infected because their parents or other care-givers have infectious cases of TB; others become infected during visits to their parents' native country.

Reporting Cases

Tuberculosis is a reportable disease in Rhode Island and throughout the United States. In addition, positive PPD results in children younger than six years of age are reportable, as a sentinel indicator of disease transmission from an adult. Timely reporting of suspect cases of TB is essential to the success of surveillance and control efforts, because prompt initiation of recommended multi-drug therapy and follow-up evaluation of exposed contacts are key to minimizing the period of infectiousness and arresting transmission.

All reported cases are investigated to assure that a definitive diagnosis is made, that appropriate treatment is initiated, and that infectious cases are quarantined during the period of infectiousness. Case data are computerized for analysis in Rhode Island. A subset of case data, stripped of names and addresses, is transmitted to the CDC as Rhode Island's contribution to national surveillance.

The TB Clinic

The State-sponsored TB Clinic is located on the campus of the Roger Williams Medical Center in Providence. The TB Clinic, with approximately 6000 patient visits/year, provides care for 98% of the active cases in the state. In addition, the TB Clinic provides evaluation and treatment for individuals with latent TB (TB infection), primary prophylaxis for TB exposure, and completion of immigration requirements for TB screening initiated overseas. Open Monday through Friday, the clinic schedules morning, afternoon and evening appointments. Patients are scheduled on a priority system that allows supsect active cases to be seen within 24 hours by physician telephone request.

Although clinical care is the first priority of the TB clinic, the site also acts as an academic teaching venue with ongoing research. Both the TB Clinic and the RI TB Control Program are recognized nationally, resulting in utilization of those sites for CDC pilot projects.

The DOH public health nurse runs a TB screening clinic where tuberculin skin testing is performed every Tuesday morning, with readings performed every Thursday morning at the TB Clinic.

Telephone consultation with the medical director of the TB Clinic, Dr. E. Jane Carter, is available to all health care providers by calling the TB Clinic.

Care at the TB Clinic is supplied to all Rhode Island residents with no out-of-pocket charges. (If patients have third-party insurance, the insurer is billed.)

Private physicians may also utilize the DOT program for patients not treated at the TB Clinic. Given TB's public health threat, consultation with the TB Clinic director is encouraged for patients treated outside of the TB Clinic setting.

A list of phone numbers follows. The TB Control Program (surveillance and epidemiology at the DOH, DOT services from the DOH, clinical referral and consultation, and mycobacterial lab services) encourages interaction with the health care providers of Rhode Island in the fight against TB.

- TB Clinic (appointments and expert consultation):456-2447
- TB Clinic location: 877 Chalkstone Avenue, Providence
- DOT services: 222-2577
- Outreach Coordinator: Mr. John Veazy
- Mycobacteriology Laboratory: 222-5587

Edward F. Donnelly, MPH, is Epidemiologist, Tuberculosis Prevention and Control Program, Rhode Island Department of Health.

Thomas Bertrand, MPH, is Chief, Office of Communicable Diseases, Rhode Island Department of Health.

Utpala Bandy, MD, MPH, is Assistant Medical Director, Division of Disease Prevention and Control, Rhode Island Department of Health, and Rhode Island State Epidemiologist.

E. Jane Carter, MD, is Clinical Assistant Professor of Medicine, Brown University School of Medicine, and the Medical Director of the TB Clinic of RI.

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Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	Oct. 1998	12 Months Ending with Oct. 1998		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	242	3,140	317.1	3,773.5
Malignant Neoplasms	201	2,495	252.0	6,955.5**
Cerebrovascular Diseases	50	615	62.1	721.5
Injuries (Accident/Suicide/Homicide)	30	361	36.5	6,963.0
COPD	33	456	46.1	402.5

Vital Events	Reporting Period		
	April 1999	12 Months Ending with April 1999	
	Number	Number	Rates
Live Births	935	13,463	13.6*
Deaths	767	9,685	9.8*
Infant Deaths	(5)	(89)	6.6#
Neonatal deaths	(3)	(65)	4.8#
Marriages	456	7,505	7.6*
Divorces	215	3,292	3.3*
Induced Terminations	407	4,770	354.3#
Spontaneous Fetal Deaths	101	1,139	84.6#
Under 20 weeks gestation	(97)	(1,056)	78.4#
20+ weeks gestation	(4)	(83)	6.2#

**Excludes one death of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 990,225

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

* Rates per 1,000 estimated population
# Rates per 1,000 live births

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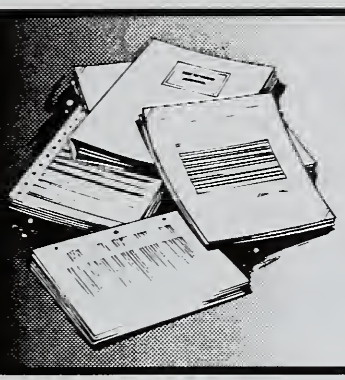

CLINICAL TRIALS DIRECTORY

Medicine & Health/Rhode Island is pleased to publish this Directory. If you would like to list a trial, please contact: Joan Retsinas, Managing Editor.
phone: (401) 272-0422
fax: (401) 272-4946
e-mail: JRetsinas@aol.com.

*** The Effect of Riluzole on the Progression of Parkinson's Disease in Patients Treated with L-Dopa or Dopamine Agonist***
Sponsor: Rhône-Poulenc Rorer
Purpose: To demonstrate that riluzole slows the progression of Parkinson's Disease by showing that treatment with riluzole delays changes in symptoms that necessitate changes in treatment.
Investigators: Joseph H. Friedman, MD, and Hubert H. Fernandez, MD
Patients Recruited: Individuals who have had Parkinson's Disease for less than 5 years and have been treateed for less than 3 years with L-Dopa or a dopamine agonist.
Intervention: Riluzole 100 mg or 200 mg per day or placebo
Duration: 1-2 years
Phase: III
Site: 130 centers throughout the world. Locally at: Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860
Contact: Margaret C. Lannon, RN, MS, phone: (401) 729-3750

*** Developmental Manifestation of Daytime Sleepiness***
Sponsor: National Institute of Nursing Research
Purpose: To examine the nighttime sleep and daytime behavior of children and adolescents on different sleep schedules
Investigators: Mary A. Carskadon, PhD
Patients Recruited: Healthy children who sleep well from 6-12 years of age
Intervention: None
Duration: 2-3 weeks
Site: E.P. Bradley Hospital Sleep Research Laboratory (located on campus of Butler Hospital)
Remuneration for Patients: $75 - $150, plus savings bond, depending on age
Contact: Gahan Fallone, PhD, phone: (401) 421-9440; fax: (401) 453-3758; e-mail: info@gahan.com

A Randomized , Double-Blind, Placebo-Controlled Study to Determine the Safety and Efficacy of FK960 in Patients with Mild to Moderate Probable Alzheimer's Disease
Sponsor: Fujisawa Research Institute of America, Inc.
Purpose: The purpose is to assess the safety and efficacy of four different doses of FK960 vs. placebo in patients with mild to moderate probable Alzheimer's Disease and to identify at least one dosage level of FK960 effective in improving symptoms of Alzheimer's Disease
Principal Investigator: Brian R. Ott, MD
Patients recruited: Patients 55 years or older with probable Alzheimer's Disease who are presently not receiving drug treatment for Alzheimer's Disease and who are not presently taking antidepressants or tranquilizers
Intervention: FK960 or placebo
Duration: 6 months
Phase: II
Site: Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860
Contact: Michael Pimental, phone (401) 729-3752

"A Multi-Center, Placebo-Controlled Trial of Melatonin for Sleep Disturbance in Alzheimer's Disease*
Sponsor: Alzheimer's Disease Cooperative Study
Purpose: Brian Ott, MD, is conducting this trial to treat Alzheimer's Disease patients who experience sleep disturbances associated with Alzheimer's Disease. Two doses of melatonin will be used to treat sleep disturbances in order to lessen the burden on caregivers and family members.

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CONTACT MELISSA SCITTARELLI AT (401) 233-2880**

Patients Recruited: Anyone 55 years or older with a diagnosis of probable Alzheimer's Disease experiencing sleep disturbances.
Intervention: Two doses of Melatonin vs. placebo
Duration of study: 12 weeks
Phase: III
Site: Alzheimer's Disease & Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860
Contact: Michael Pimental, phone (401) 729-3752

Earlier vs. Later Levodopa in Parkinson's Disease (ELLDOPA)

Sponsor: NIH and the Parkinson Study Group
Purpose: Joseph Friedman, MD, is conducting this trial to compare the effect of early or late treatment of Parkinson's Disease with L-Dopa to answer the question of whether L-Dopa slows or hastens the progression of Parkinson's Disease.
Patients Recruited: Patients must be 30 years or older, must be diagnosed with Parkinson's Disease within the last 2 years. No eldepryl, L-DOPA, amantadine, anticholinergics, antihistamines, antidepressants, or benzodiazepines for previous 30 days.
Intervention: varying doses of Carbidopa/Levodopa vs. placebo
Duration of study: 9 months
Site: Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860
Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

A Phase III Multicenter, Double-Blind, Parallel-Group Placebo Controlled Study of the Effect of Riluzole 50 mg BID or 100 mg BID for Two Years on the Progression of Parkinson's Disease in 1050 Patients

Sponsor: Rhône-Poulenc Rorer
Purpose: The purpose is to treat recently diagnosed patients with the drug Riluzole, in order to study the effect this drug has on delaying the progression of Parkinson's Disease.
Principal Investigator: Joseph H. Friedman, MD
Patients Recruited: Patients must have recently diagnosed Parkinson's Disease with symptoms present for no more than 3 years and must currently be receiving no medications to treat Parkinson's Disease.
Intervention: Two doses of Riluzole or Placebo
Duration of Study: 2 years
Phase: III
Site(s): Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860
Contact: Margaret C. Lannon, RN, MS; phone: (401) 729-3750

Pilot Huntington At Risk Observational Study (PHAROS)

Sponsor: Huntington Study Group (HSG)
Purpose: To observe individuals who are considred "at risk" for developing Huntington's disease (HD) because they have (or had) a parent or sibling with HD. Participants will be examined every 9 months for at least 3 years.
Principal Investigators: Joseph H. Friedman, MD, and Hubert Fernandez, MD
Patients recruited: "At risk" individuals 30 - 54 years old who have never been (and have no desire to be) tested for the HD gene, and who have no definite signs or symptoms of the illness.
Intervention: None
Duration of study: At least 3 years
Site(s): 38 centers in the United States and Canada. Locally at Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860
Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

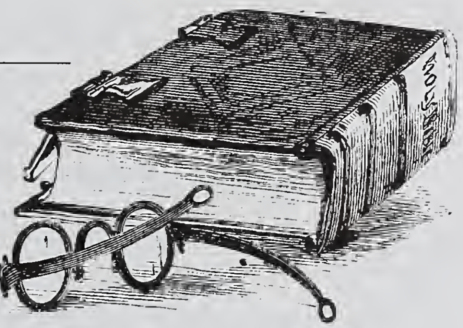
A Randomized, Double-Blind Placebo-Controlled Trial to Evaluate the Safety and Efficacy of Vitamin E and Donepezil HC1 (Aricept®) to Delay Clinical Progression to Alzheimer's Disease (AD) from Mild Cognitive Impairment (MCI)

Sponsor: Alzheimer's Disease Cooperative Study and Pfizer/Elsai
Purpose: The purpose is to determine whether treatment of persons with mild forgetfulness can prevent the actual development of Alzheimer's Disease or slow the progression of Alzheimer's Disease .
Principal Investigator: Brian R. Ott, MD
Patients Recruited: Patient must be 55 to 90 years of age and have memory difficulties that are not severe enough to interfere with carrying out one's usual daily activities (MMSE=24-30). Must be willing to take only vitamin supplement provided.
Intervention: Multivitamin, Vitamin E, or Donepezil HC1
Duration of Study: 3 years
Phase: III
Site(s): Alzheimer's Disease and Memory Disorder Clinic, Memorial Hospital of RI, 111 Brewster St., Pawtucket, RI 02860
Contact: Michael Pimental, phone: (401) 729-3752

The Effect of Pramipexole on Depressive Symptoms in Parkinson's Disease Patients

Sponsor: Pharmacia and Upjohn, Inc.
Purpose: To evaluate the effectiveness of pramipexole on the depression and other non-motor symptoms in patients with Parkinson's Disease
Principal Investigators: Joseph H. Friedman, MD, and Hubert Fernandez, MD
Patients recruited: Men and women with PD, 30 - 85 years old, who have taken no dopamine agonist medications in the past 3 months and are clinically depressed
Intervention: Pramixpexole or placebo
Duration of study: 19 weeks
Phase: IV
Site: Movement Disorders Unit, Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860
Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

Book Reviews



Skeletal Trauma

edited by Bruce Browner, Jesse Jupiter, Alan Levine, Peter Trafton

reviewed by Roy K. Aaron, MD

Skeletal Trauma has emerged as one of the definitive references on the care of fractures, dislocations and ligament injuries. Volume 1 deals with general principles, including the biology of fracture repair, analgesia, principles of internal fixation, common complications, special techniques, outcomes evaluation, and the spine and pelvis. Volume II deals with fractures of the upper extremity and lower extremity. Illustrations, photographs, and radiographs are of high quality and enhance the text materials. The chapters vary in their presentation, with some aimed at the relatively junior orthopedist and some for the more experienced. In Volume I, among the former are descriptions of the biology of fracture repair and soft tissue injuries, and the evaluation of the multiply-injured patient. Principles of non-operative, external and internal fixations are suitable for all levels of expertise. Discussions of the biomechanics of fractures and complications of fracture treatments such as compartment syndromes, regional pain syndromes, and non-union, are excellent. Somewhat more disappointing is the section on enhancement of skeletal repair. The second part of Volume I and all of Volume II deal with fractures at specific anatomic locations. For the most part, each section is authored by an individual who has made original contributions to fracture care, and the chapters reflect their expertise.

This is a multi-author book with authors chosen for their expertise in the various aspects of fracture care. It reflects the strengths and weaknesses of multi-author texts. On the positive side, opinions and reviews are generally quite expert. On the negative side, there are some unavoidable stylistic differences in organization and presentation. Overall, however, the value of expert opinion far outweighs the negative aspects. One can certainly point out favorite chapters or quibble with details; but overall this book is now a standard reference in orthopaedic trauma and should be a part of any serious orthopaedic library.

Roy K. Aaron, MD, is Clinical Associate Professor, Arthritis Surgery and Joint Reconstruction, Brown University School of Medicine.

The White Life (a novel)

by Michael Stein, MD

reviewed by James Gilcrhist, MD

When Joe Friedman asked me to review *The White Life*, the second novel by Michael Stein, my qualifications were: I had read and liked his first book, *Probabilities*; and I played basketball with Mike. *Probabilities* tells the story of a high school teenager living with his mother (his father had died a few years before), playing basketball and poker with his best friend. The poker plays a somewhat pivotal role as the two friends conspire to cheat at cards. One of the reasons I enjoyed it was that I too played a lot of poker in high school (still do) and I also cheated at it then (don't now). Beyond the analytical tools necessary to figure out a poker hand, the game is mostly about betting and bluffing, and I figured that bending the rules was just another form of appropriate misrepresentation.

The White Life is the first-person account of a few days in the life of an internist at a large academic medical center, which is by all physical resemblance, Rhode Island Hospital. Dr. Stein is a clinician and researcher at RIH and a very good one (full-time faculty at Brown, NIH funded investigator, AIDS clinic director). *The White Life* is a story with autobiographical overtones, as the professional situation of the protagonist approximates that of Dr. Stein. The plot follows the unnamed internist as he makes rounds over several days and as he prepares to confront the physician who cared for his father when he died 20 years be-

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fore, when the internist was 14 years old. A case could be made for the physician being the same character from *Probabilities*, 20 years later. There are frequent short departures as the internist examines his own motivations, and explains the workings of medicine to the reader.

It is an entertaining story, which kept my interest. It is not a thriller, a mystery or a comedy. It is, on the surface, a fairly simple story. But it really isn't so simple, as the novel works on several levels, including explaining what being a doctor is like to someone who isn't. Much of this revolves around the care of a rather cantankerous, eccentric, non-compliant and inattentive patient named George Dittus who only wants to go home. Also illustrated by the care of George Dittus is the less-than-complete grasp of the situation physicians often have, and that physicians can and do make mistakes. As one example, the physician almost kills Mr. Dittus by mistakenly administering 10 times the appropriate IV dose of an antihypertensive, and then hides the evidence. This aspect of the book helps debunk the myth of the all-knowing physician, which is a good thing; but I still cringe a bit when the flaws of physicians are exposed. Professional loyalty, I guess. I have come to view medicine as like poker in this regard. If everyone thinks his/her doctor has a straight flush, they don't necessarily need to know if all he has at the moment is 2-pair. One quote from the book, from immediately after the nearly ruinous mistake with the IV med: "I tell neither of them about the mistake, that he was close to dying because of me. I offer no awareness of what has happened and the empty vial offers no evidence against me. My reputation is strong."

The deepest level of the book examines the protagonist's need to confront the doctor who attended his father when he died. He perceives hatred for Dr. Gresser, considers him the man who killed his father, despite facts to the contrary, and after 20 years, finally reaches the moment when he can bring himself to meet Dr. Gresser face-to-face. The author gradually unveils the perplexing ambiguities that the physician confronts as he attempts to care for Mr. Dittus; the physician recognizes the same characteristics in Mr. Dittus as in his dead father. The meeting of the two doctors, one an active, aggressive, academically-oriented young internist, the other a retired, kind, Marcus Welbyesque general practitioner who worked out of an office in his house, is superficially unsuccessful but allows the main character to reach a subdued catharsis. The novel ends with George Dittus home from the hospital, and the internist glad to be home with his very patient wife and making plans to paint his house.

The White Life is well written, tells an engaging story and has a realistic plot. It is straightforward, contains no fancy verbal tricks, clunky twists, or contrived dialogue. Dr. Stein writes in a smooth, crisp manner which seems to saunter along. I enjoyed it.

James Gilcrhist, MD, is Professor in the Department of Clinical Neurosciences, Brown University School of Medicine, chief of the neuromuscular unit, and director of the EMG lab at Rhode Island Hospital.

THE RHODE ISLAND MEDICAL JOURNAL

The Official Organ of the Rhode Island Medical Society
Issued Monthly under the direction of the Publications Committee

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NINETY YEARS AGO

[OCTOBER, 1909]

In discussing "Phobias, or Fear of Disease," John B. Huber, MD, explained some common phobias. For agoraphobia, he posited biological roots: "our forbears...were arboreal in their habits, and in this lay their salvation from extinction. Comparatively feeble in body, without either weapons or defensive armor or means of concealment, they were safe only by means of the agility with which they could climb out of reach and hop from bough to bough and from tree to tree" [helped by a prehensile member]. He traced ailurophobia (fear of cats), which culminated for some patients in "catmares" and "cat terror," to allergies and heredity. He blamed pathophobia (fear of diseases) on the literature of "mercenary...quacks." He had no explanation for claustrophobia. But for the rampant fear of consumption (phthisiophobia), which caused Americans to shun anybody they suspected to be tubercular, he blamed a general ignorance. He listed the effects of phthisiophobia: a Cuban immigrant was deported from Ellis Island; neighbors opposed a dispensary for tuberculosis in New York City; Long Islanders blocked with force the landing of cholera patients headed to a treatment facility on Fire Island. Leprosy too sparked hysteria: when immigration officials tried to transport a Russian girl from Buzzards Bay to New York City for deportation, they struggled to find a schooner willing to take her. Huber predicted that, "when wisdom [comes], pathophobias will disappear."

Herbert Partridge, MD, in "Interruption of Pregnancy," listed the maternal and fetal considerations that would propel a physician to interrupt a pregnancy, whether early in the pregnancy, before the fetus was viable, or later, when the physician might induce premature labor; e.g., toxemia (the most common), eclampsia, severe vomiting, contracted pelvis, placenta previa, chorea, tumors, and a history of fetal deaths. Tuberculosis was often not a threat to the pregnancy.

FIFTY YEARS AGO

[OCTOBER, 1949]

George C. Heitzman, MD, surveyed "A Review of the Treatment of Peripheral Arteriosclerotic Disease," from care of the feet ("loose bed socks") , to intra-arterial infusion of histamine, to intermittent venous hyperemia, to lumbar sympathectomy.

Roswell W. Phillips, MD, Frederic W. Easton, MD, and Marshall N. Fulton, MD, presented "A Case of Spontaneous Rupture of the Aortic Valve" - a case with "no apparent direct precipitating factor...although due to language barrier a ...history was obtained from the patient with difficulty." The 52 year-old Italian-born hospital laundry worker developed a stomach ache and dizziness. He was admitted to Rhode Island Hospital, "with a presumptive diagnosis of hypertensive cardiovascular disease with acute left ventricular failure, and aortic regurgitation of a severe nature possibly due to a ruptured cusp." After 38 days he was discharged. For 10 weeks he felt fine at home, then suffered auditory hallucinations and lost time/place orientation, especially at night. Re-admitted to the hospital, he died. The pathologist found a ruptured aortic valve.

David S. Howell, MD, in "Methyl Salicylate Poisoning," described the case of a 66 year-old male laborer in a rubber factory who had an arthritic pain in his left shoulder. On his bedside table he kept the oil of wintergreen he used as a linament, as well as milk of magnesia. By mistake he drank the former, but recovered. Howell found 59 deaths from ingestion of methyl salicylate in the literature, starting in 1852. Most were accidental.

TWENTY FIVE YEARS AGO

[OCTOBER, 1974]

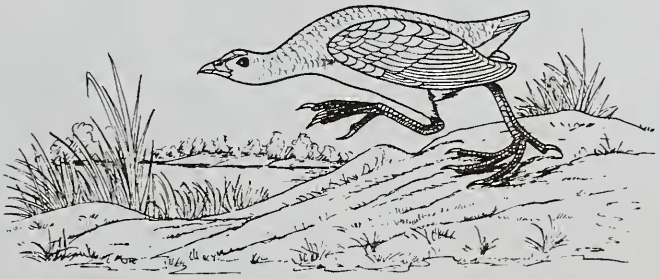
This issue included papers read at a seminar on the collaboration between pediatricians and clinical psychiatrists, geared toward improving child health care in the state. The sponsors were the Rhode Island Chapter of the American Academy of Pediatrics, the Rhode Island District Branch of the American Psychiatric Association, and the section of Human Growth and Development at Brown University.

Hector Jaso, MD, discussed "The Problem," urging flexibility: "The child and the family (often the teacher, also) are invited to tell their own stories in their own way, a truly radical departure from being just dosed and discharged." He argued against expedience, shortcuts, and "always using the same method no matter what the problem."

Mario Vigliani, MD, in "The Pediatrician Views Childhood Behavior Problems," summarized the limited community resources: "a dozen child psychiatrists (half members of the American Academy of Child Psychiatry), some mental health clinics, not designed primarily for children" with long waiting times for appointments. He cited the "tremendous numbers of demands for referral to the Child Development Center at Rhode Island Hospital" as evidence of the need for more resources.

Gunnar Nirk, MD, asked, "Why are Mental Health services for Children Inadequate?" After providing psychiatric consults to local pediatricians for 9 months, he concluded, "There are not enough funds and personnel in this community to offer treatment to all the children with serious psychiatry problems." He highlighted unhelpful professional attitudes; e.g., conviction that "it's only a stage;" preoccupation with etiology (a learning problem or a behavior problem); belief that "all s/he needs is love and understanding;" and the certainty that "to help the child we need to change the parents."

The Division of Public Assistance of the Rhode Island Department of Social Welfare published the fees they paid to physicians: $2 for an office visit, $3 for a home visit (fees lower than those recommended by the Rhode Island House of Delegates).



Information for Contributors

Medicine & Health/Rhode Island welcomes submissions from members of the Rhode Island health care community. Submissions can fall into one of four categories:

CONTRIBUTIONS

Contributions should report on an issue of interest to clinicians in the state: new research, treatment options, collaborative interventions, review of controversies. The maximum length is 2500 words; the maximum number of footnotes is 15. (The Journal is not the venue for an exhaustive literature review). Tables, charts, and figures should be camera-ready. Photographs should be black and white. (Slides are not accepted).

CREATIVE CLINICIAN

Clinicians are encouraged to submit brief (no more than 1200 words) descriptions of cases that defy texbook analysis. Photographs, charts and figures may accompany the case; footnotes should not exceed 6.

IMAGES IN MEDICINE

Clinicians are encouraged to submit unusual images (e.g., MRI, X-RAY, Ultrasound, CT), with brief (no more than 300 words) discussions.

POINT OF VIEW

Readers are invited to share their perspective on any issue facing clinicians. The topic is broad; e.g., ethics, health care policy, and/or relationships with patients. Maximum length is 1200 words.

The format of submissions is as follows:

The title page should include name, affiliation, address, phone, fax, and e-mail. A brief abstract should appear on a separate page. References should be numbered sequentially in the text and listed separately at the end of the document (not the end of each page).

FOR CONTRIBUTIONS, IMAGES IN MEDICINE, and POINT OF VIEW, please submit 4 hard copies of the document, with a disk (Microsoft Word or Text), to the managing editor: Joan Retsinas, PhD, 344 Taber Avenue, Providence, RI 02906.

FOR CREATIVE CLINICIAN columns, please submit 3 hard copies of the document, with a disk (Microsoft Word or Text), to Anthony Mega, MD, Miriam Hospital, Providence, RI 02906.

For additional information, please contact Joan Retsinas (phone: (401) 272-0422; fax: (401) 272-4946; e-mail: JRetsinas@aol.com).

BOOK REVIEWS

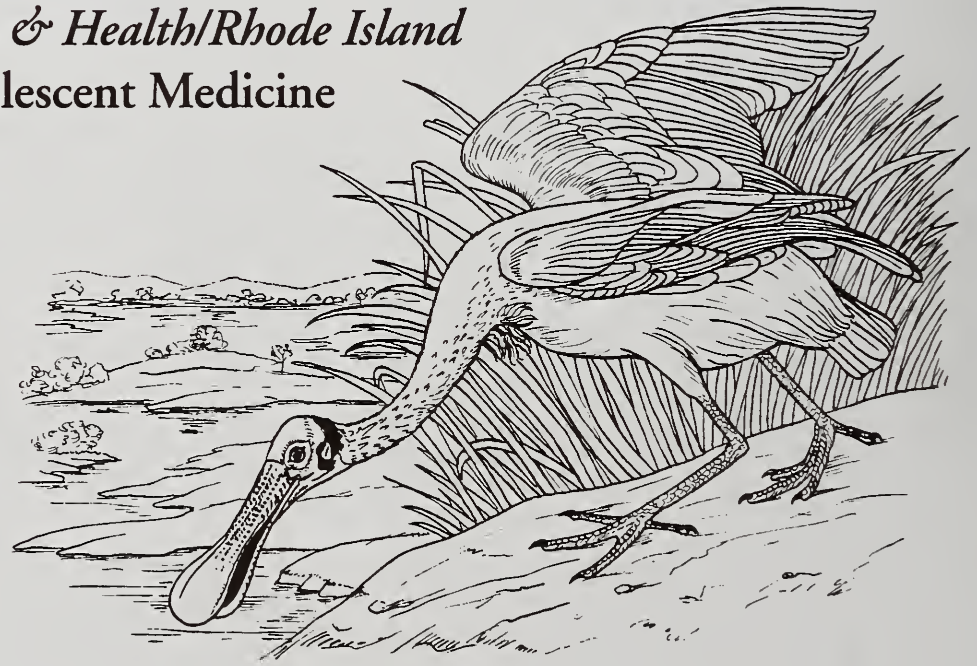
Medicine & Health/Rhode Island will review books authored by Rhode Island physicians. Publishers should send a copy for review to the managing editor.

Forthcoming ...

NOVEMBER

Medicine & Health/Rhode Island

Adolescent Medicine



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Medicine and Health
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COMMENTARIES

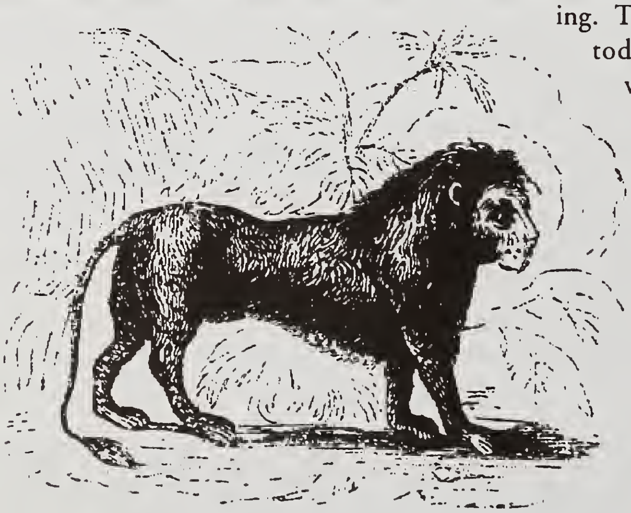
Omerta

A new book, *Blind Eye*, by Pulitzer Prize author James B. Stewart tells the story of Michael Swango, MD, a convicted poisoner, fraud and alleged serial killer. It is a riveting, muckraking account that really tells two quite distinct tales. One describes what a smart psychopath can accomplish, in this case possibly 60 homicides. The other is the equally horrifying description of a medical establishment unable and unwilling (Ohio State University primarily) to deal with one of its own when faced with behavior it hopes to wish away by turning a blind eye (hence the title).

My own interest lies in the choice that various medical establishments made to not investigate this ingratiating, hard-working psychopath. Don't we believe that the medical profession holds itself to a higher standard than other professional guilds and assume that our peers hold to these standards? Yet it is rare, for example, for medical schools to drop students, which is not because admissions criteria are so strict. We don't like to admit mistakes, and also by the time problems emerge the student has invested a huge effort. I suspect too that we feel a serious problem will be dealt with later, in training. And to some extent this is true.

Training programs do expel resident physicians. This is uncommon and almost always for personality/behavioral issues. Poisoning patients would surely be considered such a problem but it is so bizarre, so inconceivable that Dr. Swango apparently did get away with murder.

How should we respond to bad medical or ethical practices? Where I trained, and probably in most hospitals in this country, we had "single 0" (licensed to maim) and "double 0" (licensed to kill) doctors. Bad doctors with largely Jewish practices were said to have "killed more Jews than Hitler." A patient was "aitched" if Dr. H. had another surgical mishap. All of these statements represented black humor and not true malpractice, but there is often a touch of truth in these types of rumors. Substandard care, malpractice and malfeasance are, however, quite different. Twenty years ago a colleague in training was arrested and convicted of pederasty in New York. Evidently, his plea bargain allowed him to continue practicing medicine in adult-only facilities, since he continued practicing. This could not happen today in Rhode Island where a vigorous board of licensure and discipline oversees medical malfeasance.



My experience with unacceptable medical practice in Rhode Island has been mixed but has improved. A case of physician incompetence recently reported to a hospital led the medical staff to refer the case to the state for possible license revocation or other action. Yet, the Belasco case occurred in R.I. a decade ago. Dr. Belasco, working at a community hospital, inserted more pacemakers of a certain type than the rest of New England put together. He had falsified records, even using other people's EKGs to justify the procedures. How this could continue undetected for years was never reported in the newspapers. Possibly, as in the Swango case, the nurses and other non-MD professionals were not listened to and feared the retribution that so often plagues the whistle blower. Probably the physicians lacked proof and were therefore inhibited, or perhaps could not believe their own eyes that such a thing could happen.

A surgeon implicated in excess post-operative infections was shielded by the infection control unit because upper-level administrators did not want to antagonize an important hospital donor/fundraiser.

A chair of a department at Brown refused to put into writing his negative statements about a doctor applying for privileges at a Brown-affiliated hospital, presumably for fear of being sued.

I recall reading of a case in upstate New York where a physician enrolled in a residency program after favorable letters of recommendation were received despite the fact that the referees knew the resident had been convicted of rape and failed to mention it. The authors of the letters of reference were found culpable in court. I once interviewed a candidate for a position who had an excellent CV. It was unclear, however, why he left a distinguished university to work in a seemingly undesirable, undistinguished hospital in a part of the country where he had no friends or relatives. In passing, he mentioned a TV news show that he was almost on. He had been accused of fraudulent research and had lost his ability to ever obtain government research funding. Was this in his letters of recommendation? No. Yet when I called his former chair, the issue was well known and I was cautioned about hiring him.

Rhode Island's board of licensure and discipline stands in contrast to Ohio State (as presented in the book). Yet the board's efficacy rests on our monitoring ourselves. They don't investigate without complaints.

The medical guild has a special place in society. The entrance to one hospital I know bears the inscription, "For of the most high comes healing." Yet our own peer review is often too shallow to maintain this status. If we believe it is important to police ourselves, then we must exercise the utmost vigilance. We must maintain our high standards. We cannot maintain a "blue wall of silence" like the police, or the "omerta" of the mafia. We cannot assume that all's well simply because a physician says so. In *Blind Eye* we see the result of our failures in a worst case scenario.

– Joseph H. Friedman,MD

A General Practitioner Confronts the White Plague

Progress in science, more often than not, is an incremental thing made up of the collective contributions and insights of many. Rare indeed is the scientist, working in heroic isolation, who discovers a major cure. More often, the creative scientist critically reviews the prior publications of others and then builds upon this structure. Indeed, if there is one factor uniting the sisterhood and brotherhood of research workers it is the precious existence of a vigorously critical, open, transnational scientific literature.

Of course it takes a measure of courage to publish in a medical journal since one's contribution will then be analyzed mercilessly by peer scientists. Occasionally, though, a scientific publication commands the immediate awe of the fussy readership, offering observations so significant that they stand out as definable advances in humanity's struggle against disease. Such a paper was Robert Koch's publication on the etiology of tuberculosis appearing in the April 10, 1882, issue of Berlin's weekly medical journal [*Berliner Klinische Wochenschrift.*]

Koch was born in 1843 near Hanover, Germany. He received his medical education at Gottingen University, where he was influenced by the brilliant teaching of Jacob Henle. Koch established a general practice in Wollstein but from the beginning was drawn to the newly evolving science of bacteriology. In 1876 Koch announced that he had grown the anthrax bacillus in pure culture. Emboldened by this initial laboratory success he relinquished his practice and accepted a position in Berlin's Institute of Health.

Medicine was beginning to learn the malevolent role of bacteria in human disease, but little had yet been done to distinguish, by microscopy, one germ from another. Some bacteria were small and rounded [the cocci] and some elongated [the bacilli] but little else was known to distinguish the many varieties of germ. The leading biologist of the day even lumped all bacteria into one genus which he called Chaos. It was in such a primitive state of microbiological understanding that Koch began his research efforts to explore the cause of the most prominent disease of his day, tuberculosis.

When Koch began his labors, tuberculosis, sometimes called the white plague, caused the death of about one out of three European adults. Its cause was unknown and many in the clinical community resisted the idea that it was a communicable process despite the demonstration by Villemin in 1865 that the human disease could be experimentally transmitted to rabbits and thence serially passed to yet other experimental animals. And even the very definition of the disease remained unsettled. Many physicians claimed that scrofula, tuberculosis, consumption and phthisis were separate disorders.

Koch's 1882 publication boldly announced three major discoveries regarding tuberculosis.

Prior research workers, using conventional staining procedures, had failed to distinguish any germs within affected tuberculous tissues, leading them to conclude that microbes played no part in tuberculosis. Ehrlich had previously noted that certain aniline dyes clung selectively to some bacteria and might therefore be used to make germs selectively visible under the microscope. Koch now adopted this principle by employing a phenyl dye [called

Bismarck Brown in honor of the Prussian leader] and abundant numbers of rod-shaped germs [bacilli] suddenly appeared in his microscopic preparations of tuberculous tissue. A simple modification of a standard procedure demonstrated the heretofore hidden organisms. Koch called them the tubercle bacilli and demonstrated them in all the diverse forms of human consumption. Koch declared that tuberculosis had many clinical faces but only one underlying cause. Furthermore, he showed that many emaciating diseases of chickens, pigs and cows [including a widespread disease called Perlsucht] were all pathological manifestations of the same tubercle bacillus. Koch's observations represented more than a unifying biological concept; they carried immense public health implications.

Koch's second series of experiments concerned methods of culturing his newly discovered bacillus. After many trials he found that sterile, coagulated sheep serum provided an ideal medium for the laboratory growth of the organism. Having demonstrated an infectious agent in tuberculosis and knowing that the disease was predominantly within the lungs, Koch then speculated that human sputum was the vehicle by which the disease spread from person to person. Accordingly he cultured the expectorations of numerous consumptive patients and readily recovered his organism from each. This, too, had public health implications. Spitting was not only unesthetic; it also constituted a major pathway for the spread of tuberculosis. Public education programs were then established and spitting in public became a crime.

The third series of experiments in Koch's paper was, in some ways, the most crucial. He injected the bacterial colonies growing on his special culture medium into healthy animals; and within weeks these animals developed typical tuberculosis.

Thus, in a single brief paper, Koch demonstrated a unique organism invariably associated with all phases and clinical variants of tuberculosis; devised a means by which these organisms might be grown in test tubes; and then demonstrated that these virulent organisms, after successive subcultures, caused tuberculosis in healthy animals. Koch was then justified in claiming that he had discovered the cause of tuberculosis thus fulfilling the earlier contention of his teacher, Henle, that the coexistence of certain bacteria in diseased tissues did not prove causality but was merely the first element in such a proof.

Koch's corollary demonstration of tuberculosis affecting the udders of many dairy cows explained the origins of numerous cases of intestinal tuberculosis in children and led to animal inspection and sterilization programs of bottled milk.

Koch thus identified the causal agent of a major disease. Furthermore, he outlined ways of preventing its spread within the community: by isolating patients with active tuberculosis, by educating the public concerning the morbid role of sputum, and by sterilizing the milk supply. The adoption of these measures resulted in a notable decline in the frequency of tuberculosis. Koch's later accomplishments included the discovery of the cause of cholera; but when he was awarded the Nobel Prize in 1905, it was his 1882 paper on tuberculosis which was specifically cited.

– Stanley M. Aronson, MD

Adolescent Medicine

Adolescent Medicine addresses the health needs of young people in the second decade of life, a period of rapid physical and psychologic growth and development. Although perceived as one of the healthiest periods in the life cycle, adolescence has its special health concerns, vulnerabilities and barriers to health care. Over the past 20 years, the adolescent death rate has declined. In the 1930s approximately 75% of adolescent deaths were due to natural or physical causes. Today, 75% of adolescent deaths are due to personal behaviors and hence are preventable. In the United States the three major causes of adolescent mortality are motor vehicle accidents, homicide, and suicide. Factors associated with adult morbidity and mortality include poor nutrition, substance abuse, a sedentary lifestyle, unsafe sexual practices, and risky motor vehicle behaviors. These behaviors begin in the adolescent years. Thus, in addition to treating physical disease and the sequelae of these behaviors, we need to actively attempt to prevent or modify these health risk behaviors.

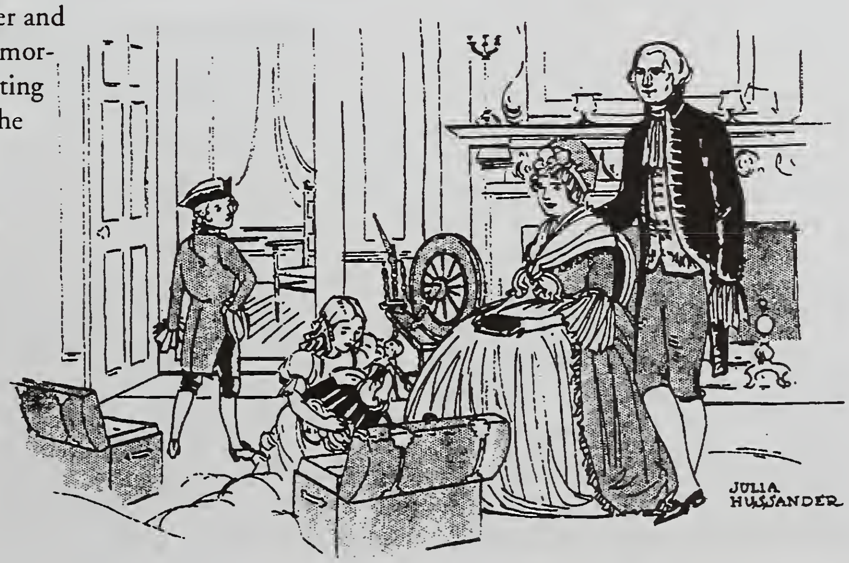
Preventive services for infants and children are the mainstay of pediatric primary care. Why then do we provide sporadic, preventive services for our adolescent patients? Several factors are involved, including lack of provider knowledge of preventive health guidelines, lack of adequate reimbursement for what is often a time-consuming visit, and a lack of access to care.

Clinical preventive medicine in the 1990s has fortunately become an attractive means of curbing escalating medical costs, and several preventive service initiatives have been developed. Guidelines for Adolescent Preventive Services (GAPS) offers a package of clinical preventive services that is tailored to the adolescent population, is practical to implement, and supports the reduction of adolescent health risk behaviors. It may be obtained from the American Medical Association (phone: (800) 621-8335).

This issue highlights medical and psychosocial health conditions in adolescents. The articles point out that the approach to prevention, diagnosis and treatment of these conditions in adolescents must take into account their physical and psychological developmental stage.

Adriana Schmidt describes the growth of adolescent medicine as a discipline, reflected in the development of specialized adolescent clinics and in the formation of the Society for Adolescent Medicine, a professional organization interested in providing health services to adolescents, conducting research in this age group, and training professionals in this field. Using cardiovascular disease, hypertension and osteoporosis as examples, Dr. Harel discusses the importance of optimizing the nutrition of adolescents. Dr. Riggs' article on eating disorders in adolescents is a good example of the age-specific impact of starvation on pubertal growth, menstrual function and bone density.

We are all frustrated by the inability to access mental health services for our patients. This is especially problematic in adolescents, where suicide is the leading cause of death. Dr. Kazura suggests how to curb this alarming statistic by outlining the steps for routine screening of depressive symptoms, education of patients and their families, and psychiatric referral. Both cancer and cardiovascular disease account for significant morbidity in adolescence. Dr. Ferguson's interesting case discussion of an 11 year-old boy with the common symptom of muscular pain reminds us to be vigilant for the possibility of malignancy. The sudden death of a young athlete is a sad and frightening event and is usually linked to a previously unknown cardiac condition. By giving us an organized approach to the diagnosis of syncope in the adolescent, Dr. Feit's discussion should help us feel more prepared to take care of an adolescent with a sudden loss of consciousness. Abstinence and safe sexual practices are still the best means of preventing pregnancy. Dr. Flanagan's article reminds us of the importance of discussing the "availability" of this method of emergency contraception with parents and adolescents of reproductive age whether or not they are sexually active.



Finally, an aspect of adolescent health care access is addressed by Rosemary Reilly-Chammat in her discussion of school-based health centers in Rhode Island's urban high schools and middle schools. With the recent violence in suburban schools, a discussion of school-based mental health services in these settings may be warranted. While it has been shown that school-based health centers improve adolescents' access to health care, it is unclear how these centers will continue to be financially sustained.

What is clear, however, is that practitioners in all settings need to provide preventive services for adolescents if we are going to impact on the health risk behvaiors that cause the greatest morbidity and mortality. With this approach, we can help adolescence become one of the healthiest and happiest periods in the life cycle.

– Rosalind Vaz, MD

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The History of Adolescent Medicine:
Three Periods of Awareness and Development During the Twentieth Century

Adriana Schmidt, AB

Abbreviations Used:

SAM	Society for Adolescent Medicine

For a long time, adolescence was a life stage we didn't think about. Colonial Americans and early modern Europeans distinguished "youth" from adulthood, but this stage was seen neither as a fixed age span nor as a uniform set of experiences. "Youth" was, instead, a period of gradual introduction to adult roles and responsibilities.[1]

When Stanley Hall published *Adolescence: Its Psychology and Its Relation to Physiology, Anthropology, Sociology, Sex, Crime, Religion and Education* in 1904, the "child study pioneer and educational reformer" introduced Americans to the idea that adolescence should be considered a distinct developmental category. His two-volume work also showed that during adolescence, somatic and psychological growth functioned synchronously. In what became the basis for the twentieth century concept of youth, adolescence was not a vaguely defined time of gradual change but a period marked by dynamic changes in biological and emotional processes.

Hall's idea that adolescents shouldn't be forced to conform to adult standards influenced numerous progressive era reforms of education and parenting. Yet *Adolescence* was also published at an opportune moment in the history of medicine: pediatrics was becoming more specialized. After the turn of the century, pediatricians became more interested in incorporating a behavioral component into the study of human development in early life.

Despite these interests, the field of adolescent medicine only emerged in the 1950s, largely because of J. Roswell Gallagher. Like pediatricians before him, Gallagher was interested in studying adolescent health from both a physical and behavioral perspective. However, he was the first physician to argue that adolescents have health care needs that are separate from those of both adults and children, and should be seen by doctors sensitive to those needs.

Between the 1940s and the early 1960s, Gallagher laid the structural foundation for modern adolescent medicine. In 1937, he launched the "Grant Study," in which he looked at the relationship between somatotype and personality in teenaged boys. Charles Janeway, a physician-in-chief at Boston's Children's Hospital, was concerned about the lack of training in the psychological aspects of childhood health and disease in residency programs. He recognized Gallagher's efforts to study the adolescent from a variety of disciplinary perspectives. In 1951, Gallagher and Janeway submitted a proposal for a new Adolescent Unit at Boston Children's to the board of trustees. By the mid-1950s, twelve fellowship positions were available in adolescent medicine.

> *Currently there are approximately 1400 members in the Society for Adolescent Medicine...*

The hallmark of Gallagher's work was his recognition that, though the problems facing adolescents were not often life-threatening, they could be devastating to their emotional well-being. This led him to establish a revolutionary patient-physician relationship in which adolescents were seen separately from their parents and were considered more responsible for their own health. More importantly, Gallagher felt that physicians needed to consider the health of the whole patient rather than focus solely on an adolescent's particular complaint.

By the late 1960s, several adolescent medicine specialists saw the importance of granting more autonomy to young adult patients. The relevance of confidentiality was enhanced by the national problem of sexually transmitted diseases and pregnancy among teenagers, and consent laws changed in most states by the mid-1970s. A rise in the number of free clinics across the country as well as hospital-based clinics and adolescent inpatient wards also marks a subsequent movement to cre-

ate new systems of medical care that welcomed teenagers. Between 1968 and 1989, specialized physicians expanded their services and aimed to improve their approach to adolescents. The field of adolescent medicine also became more professional as it slowly came to be recognized as a medical subspecialty.

In 1968, the Society for Adolescent Medicine (SAM) formed, with Dr. Gallagher as president. The group sought to define the boundaries of adolescent medicine; to establish standards for education and training; to encourage medical students and residents to consider a career in adolescent medicine; and to investigate adolescent health problems and offer medical consultation for such problems on a national level. Ten years later, the American Academy of Pediatrics established a Section on Adolescence, with the hopes of integrating adolescent health programs into all pediatric residency programs. In 1978, the Task Force on Pediatric Education also helped adolescent medicine gain a stronger foothold in medical school and pediatric residency training. Finally, in 1983, non-physicians could become members of SAM, though 90% of the members were still physicians by the mid-1980s.

The 1980s saw debate about whether adolescent medicine should formally become a subspecialty. In 1987, SAM president Joe Sanders appointed a subspecialty committee to write up a proposal for the American Board of Pediatrics, which was submitted in 1989. The first board examination took place in 1993, formally recognizing adolescent medicine as a subspecialty.

Currently there are approximately 1400 members in the Society for Adolescent Medicine and 380 American physicians are board-certified in adolescent medicine. These physicians continue to teach adolescent medicine to residents in pediatrics, internal and family medicine. Hasbro Children's Hospital houses the only Division of Adolescent Medicine in Rhode Island: it just celebrated its sixteenth anniversary.

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Adriana Schmidt graduated from Brown University in 1999. This article is based on her honors senior history thesis, "American Girls and the Gynecologic Exam: The History of Pediatric and Adolescent Gynecology."

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New Developments in Adolescent Nutrition

Zeev Harel, MD

An extensive body of scientific evidence indicates that the typical Western diet, high in total fat, saturated fat, and sodium, and low in complex carbohydrates and fiber, is associated with obesity, heart disease, and some forms of cancer. In 1991, recognizing the crucial role of diet and nutrition in disease prevention, in 1991 the US Department of Agriculture and the Department of Health and Human Services proposed the 2000 objectives for health promotion and disease prevention, with a major emphasis on children's and adolescents' nutrition.[1] To achieve these goals the Food Guide Pyramid was developed in 1992.[2] This guideline translates nutrient intake into appropriate servings from five major food groups (grain, vegetable, fruit, dairy, meat), and is taught in health classes during the 6th or 7th grade. Recent data indicate, not surprisingly, that our teens' eating patterns do not meet national recommendations.[3] In this article, we discuss new nutritional data which have implications for the present and future well-being of adolescents.

Abbreviations Used:

DHA	docosahexaenoic acid
DPA	docosapentaenoic acid
EPA	eicosapentaenoic acid
GAPS	guidelines for adolescent preventive services
HDL	high-density lipoprotein
LDL	low-density lipoprotein
LNA	linolenic acid
NIH	National Institutes of Health
PUFA	polyunsaturated fatty acid
RDA	recommended dietary allowance
VLC	very long chain

PREVENTION OF CARDIOVASCULAR DISEASE

There has been a debate among scientists about how early in life dietary changes must be implemented to reduce the risk of heart disease later in life. Studies based on autopsies of male soldiers killed during the Korean and Vietnam wars found a substantial amount of atherosclerotic plaques in these young, apparently healthy, men. Strong reviewed the data and found that serum cholesterol and elevated blood pressure were consistently and positively related to the degree of atherosclerotic lesions, while the level of high-density lipoprotein (HDL) cholesterol was inversely related to the lesions.[4] A recent study which for the first time included a large sample of young women (1079 men and 364 women age 15-34 years) showed that low-density lipoprotein (LDL) cholesterol and smoking were associated with atherosclerosis in both sexes and in Caucasians and in African Americans.[5] While the exact age of onset of atherosclerosis is unknown, it is apparent that this is a continuous process starting during the second decade of life.

Hypercholesterolemia is a well-known factor associated with the risk of atherosclerosis. Cholesterol levels in young adults have been shown to predict the development of coronary heart disease up to 40 years later,[6] and many educational programs and treatments have been developed to decrease cholesterol levels. Little is known, however, about how well childhood cholesterol levels may predict adult levels. In the Muscatine Study, some children (age 8-18 years) with high cholesterol levels had high levels of LDL-cholesterol as adults. [7] Current guidelines for adolescent preventive services (GAPS) recommend cholesterol screening during late (18-21 years) adolescence, or earlier if there is a family history of cardiovascular disease prior to age 55, or a parental history of high cholesterol. Physicians may order cholesterol screening if the family history is unknown, or if the adolescent has multiple risk factors for future cardiovascular disease.

Both the total amount and the type of fat consumed are related to the incidence of cardiovascular disease. In the past, attempts to lower saturated fat and raise polyunsaturated fatty acids (PUFAs) were found insufficient because they lowered the HDL cholesterol by about the same percentage that they lowered the LDL cholesterol. Two additional changes have been shown to improve the lipid profile: Increasing the amount of monounsaturated fatty acids (found in olive oil) was found to improve HDL-cholesterol, and increasing dietary intake of omega 3-polyunsaturated fatty acids resulted in lowering triglyceride levels, both changes leading to an ideal lipid profile.

Current fat intake of US adolescents is about 36% of energy intake, with up to 16% as saturated fat. There is a need to reduce this fat intake and to replace the amount reduced by complex (not refined) carbohydrates. However, we have to avoid overzealous restriction of dietary fat, since fat is essential for growth and pubertal development of adolescents. Ideal fat intake for adolescents should be 30% of energy intake, with 10% saturated fatty acids, 10% monounsaturated fatty acids, and 10% polyunsaturated fatty acids.

Since oxidation of LDL-cholesterol plays an important role in the pathogenesis of atherosclerosis,[8] studies have investigated a possible intervention with anti-oxidant vitamins such as vitamin A, C and E. While there are insufficient data to recommend daily supplementation with antioxidants, adolescents should be encouraged to consume at least the recommended dietary allowance (RDA) for these antioxidant vitamins.

Another recently recognized atherosclerosis risk factor is homocysteine. Increased homocysteine levels have been associated with increased risk for atherosclerosis and thrombosis in some patients.[9] Deficiencies of folate, vitamin B12, or vitamin B6 (three vitamins involved in the normal metabolism of methionine) may lead to an increase in homocysteine levels. In certain cases, ordering a test to determine homocysteine levels may be advisable, and, if elevated, treatment with these vitamins (at least folic acid) may be considered.

PREVENTION OF HYPERTENSION

Hypertension is a major contributor to the development of cardiovascular disease, stroke, and renal failure. Five micronutrients have been shown to influence blood pressure in humans:[10]

1. *Sodium:* Linear relationship between prevalence of hypertension and salt intake has been shown in populations around the world. However, clinical trials with dietary salt restriction have produced heterogeneous results, indicating that only a subset of the population is sensitive to the blood pressure-elevating effects of salt. Severe salt restriction may have negative effects on lipid profile and insulin, and may increase blood viscosity. Therefore, adolescents should be encouraged to consume between 500 mg (minimal dose) and 2400 mg (maximal dose) of sodium/day.

2. *Chloride:* Chloride without sodium does not increase blood pressure. However, administration of sodium with chloride exacerbated hypertension in animals and in humans, whereas non-chloride sodium salts showed no effect on hypertension.

3. *Potassium:* High potassium intake has been associated with lower blood pressure levels, and a diet high in potassium can offset the hypertensive effect of high sodium chloride intake. In addition, supplementation with potassium resulted in a decrease in blood pressure in some studies. Adolescents should be encouraged to increase consumption of fruits and vegetables rich in potassium.

4. *Magnesium:* Low dietary intake of magnesium may increase the risk of hypertension. Relatively high doses of magnesium (2 1/2 times the RDA) were required to lower blood pressure in patients with hypertension. Adolescents should be encouraged to consume foods rich in magnesium such as whole grains, green vegetables, and bananas.

5. *Calcium:* Inadequate calcium intake has been associated with increased blood pressure levels in humans. Increasing dietary calcium intake may prevent hypertension, and may offset the hypertensive effects of high sodium chloride intake. A modest antihypertensive response has been shown in calcium supplementation clinical trials. In particular, adequate calcium intake may prevent gestational hypertension.

PREVENTION OF OSTEOPOROSIS

Adolescents grow at the greatest rate of any age group after infancy, and accumulate about 40% of their total bone mass during puberty. Genetic and ethnic factors, dietary habits, body weight, and other lifestyle factors such as physical activity and smoking, influence the accretion of bone mass during adolescence. In particular, the growth of the skeleton requires a positive calcium balance to reach an optimal peak bone mass. Because maximum bone mass is acquired during puberty, the calcium deposited during these years may determine the risk of osteoporosis and fractures in adulthood. It is therefore conceivable that one approach to reducing the risk of osteoporosis later in life may be the provision of adequate calcium intake during the formative years. The last (1989) RDA of calcium for adolescent boys and girls was 1200 mg/day. Concerns that this amount may be insufficient prompted the 1994 National Institutes of Health (NIH) Consensus Development Conference to recommend increasing calcium intake to 1200-1500 mg calcium/day for adolescents. In 1997 the Institute of Medicine's new Dietary Reference Intakes recommended calcium intake of 1300 mg/day for adolescents.[11] The Institute of Medicine also stated that up to twice this daily amount is considered safe, and that consumption of more than twice the recommended amount may increase the risk of developing kidney stones.

At this stage of increased need for calcium, the majority (89%) of adolescents in a study in Rhode Island[12] were found to consume significantly less than the RDA for calcium, with boys consuming 681 + 28 mg/24 h (57% of RDA), and girls consuming 536 + 19 mg/24 h (45% of RDA). About one third (36%) reported daily supplementation with a vitamin and mineral preparation. However, the supplements included only between 100 and 200 mg of elemental calcium, and failed to significantly increase calcium intake in these adolescents. Most adolescents were aware of the main health benefits of calcium, but did not know about daily requirement and calcium content of the various dietary sources. Health classes were the main reported source of information about calcium. Only a third of the adolescents who participated in the study reported that their care provider had discussed the health benefits of calcium with them. Knowledge that adequate calcium intake during adolescence is critical for prevention of osteoporosis was associated with higher consumption of calcium, and adolescents who were aware of the daily requirement for calcium tended to consume more of this essential mineral. Thus, the study provides some evidence to suggest that adolescents with more knowledge about calcium consume larger amounts of the mineral.

> *With adolescents it is important to be specific.*

Adolescent health providers, nurses, dietitians, and health teachers have to provide information about the health benefits of calcium, including the role of calcium in regulation of blood pressure, and the possible role for calcium in prevention of colon cancer. In particular, we have to emphasize the important role of calcium in peak bone mass accretion during adolescence. With adolescents it is important to be specific. Concrete information about calcium sources and amounts should be provided. Adolescents should be recommended to drink 1 1/2 cups of milk in the morning and 1 1/2 cups of milk at bed time. Adolescents should also be informed about non-dairy sources of calcium such as fruit juices and cereals enriched with calcium, which have been increasingly available in recent years. When adolescents fail to consume appropriate amounts of calcium, calcium pill supplements containing 500 mg of this mineral should be considered. Since dietary habits of the family and peers, as well as advertising media, may affect adolescents' dietary habits, these factors should also be included in every calcium educational program for adolescents.

The Role of Omega-3 PUFAs in Disease Prevention and Attenuation of Inflammatory Processes

Many epidemiological, interventional, and animal studies have shown that an adequate consumption of n-3 PUFAs of marine origin may reduce the risk of cardiovascular disease, hypertension, and ischemic stroke.[13] Populations with high consumption of n-3 PUFAs have experienced low incidence of breast cancer, and it has been suggested that fat intake in childhood or adolescence might be related to breast cancer risk later in life. In addition, recent reports have suggested that increasing dietary intake of n-3 PUFAs results in formation of less potent inflammatory mediators (prostaglandins from the third series and leukotrienes from the fifth series) and attenuates inflammatory processes which may affect adolescents. Dysmenorrhea, the most common gynecologic complaint among adolescent girls, correlated with low dietary intake of n-3 PUFAs, and supplementation with n-3 PUFAs was effective in alleviating symptoms of dysmenorrhea in adolescents.[14] High dietary intake of n-3 PUFAs was associated with higher levels of pulmonary functions in patients with asthma, and dietary supplementation with n-3 PUFAs had a beneficial effect in cystic fibrosis, inflammatory bowel diseases, rheumatoid arthritis, and chronic renal diseases. However, a recent study (submitted for publication) in Rhode Island found that the majority of adolescents who reported suffering from an inflammatory disease were unaware of the potentially beneficial effect of n-3 PUFAs, and their dietary intake of n-3 PUFAs was low.

Linolenic acid (LNA) is an essential n-3 PUFA and is converted in the body to the very long chain (VLC) n-3 PUFAs eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA), and docosahexaenoic acid (DHA). Limited dietary supply of LNA and limited ability of the body to make EPA, DPA, and DHA from LNA have prompted a recommendation to increase direct consumption of VLC n-3 PUFAs which are mainly found in seafood. Fish intake of at least two to three fish meals per week has been recommended to meet the requirements for VLC n-3 PUFAs.[13] The 1990 Canadian Nutritional Board recommended a daily n-3 PUFAs intake of 1.4 gm for boys and

1.2 gm for girls age 13-15 years, with optimal intake of 800-1100 mg/d for linolenic acid and 300-400 mg/d for VLC n-3 PUFAs.[13] The current ratio of n-6 PUFAs to n-3 PUFAs in the Western diet is 20-25/1, while a century ago it was 1:1. The ideal ratio according to the Canadian Nutritional Board should be around 6:1.[13]

In conclusion, establishment of adequate n-3 PUFAs intake during adolescence may reduce the risk of major life-threatening diseases such as cardiovascular disease and hypertension later in life. Aside from preventing diseases, n-3 PUFAs may benefit adolescents by alleviating inflammatory processes, such as dysmenorrhea, as well as ameliorating inflammatory diseases such as asthma. It is therefore necessary to motivate adolescents to increase n-3 PUFAs dietary intake for their present and future health and well-being.

THE ROLE OF FOLIC ACID IN PREVENTION OF NEURAL TUBE DEFECTS (SPINA BIFIDA, ANENCEPHALY, ENCEPHALOCELE)[15]

Many studies have shown that adequate consumption of folic acid before conception may prevent neural tube defects and other congenital anomalies. While the exact mechanism of this effect is unclear, it may be related to folic acid induced decrease in homocysteine levels. To prevent the first occurrence of neural tube defects, it is recommended that all women of childbearing age should consume 0.4 mg (100% RDA) of folic acid a day. To prevent recurrence, high dose of folic acid, 4.0 mg daily (10 fold RDA) is recommended from 1 month before conception through the first 3 months of pregnancy.

Folic acid sources include: chicken liver, green vegetables (asparagus, spinach, Brussels sprouts, turnip greens), beans (black, white, navy), orange juice, and cereals. Since January 1998, all enriched grains produced in the US have been fortified with folic acid at a concentration of 0.14 mg per 100 grams of grain. For many adolescents and young adults, however, the easiest and most certain way to get adequate folic acid is to take folic acid supplements, either alone or as part of a multivitamin supplementation.

SUMMARY

Many adolescents are not aware of optimal dietary requirements and this lack of knowledge may adversely affect their present well-being and future health. Adolescent health providers, nurses, dietitians and health teachers should disseminate information about dietary requirements and about important nutrients for adolescents.

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Eating Disorders in Adolescents:
Role of the Primary Care Physician

Suzanne Riggs, MD

Although eating disorders (EDs) primarily affect adolescents and young adults, much of the recent medical and psychiatric literature fails to consider the unique physiologic, psychologic and developmental issues relevant to younger patients.

Epidemiology and Diagnostic Criteria

Incidence rates for anorexia nervosa (AN) have increased steadily from 1955 to 1984 among those 10-19 years of age, but not among adults.[1-3] The prevalence rate of AN of 0.48% among girls 15-19 years old in America makes this disorder the third commonest chronic condition among adolescent girls after obesity and asthma.[1,3] Approximately 1-5% of adolescent girls were reported in studies during the 1980ís to meet strict DSM III R criteria for bulimia nervosa (BN) while as many as 10-50% reported they participated in occasional self-induced vomiting or binge eating.[1-3] The onset of AN usually occurs in early to late adolescence but increasingly is appearing before puberty and in males as well as females. Large numbers of teenagers who have disordered eating do not meet the strict DSM-IV criteria for AN or BN. (Table 1). Patients with these "subclinical" EDs could still suffer health risks and long term sequelae even though they don't satisfy strict criteria. Patients with mixed symptoms of AN and BN or patients who satisfy most DSM criteria but are prepubertal or male are usually classified as "ED, not otherwise specified" (ED, NOS) and also require careful attention and medical follow-up.

Causes

Monozygotic and dizygotic twin studies and family studies indicate that ED have a genetic component. A woman has a 10-20 times higher risk of developing AN if she has a sibling with the disease.[2] Eating disorders are seen primarily in western and industrialized countries. Cross cultural studies, however, reveal that the incidence of these disorders is increasing in rapidly developing societies in other parts of the world.[1] EDs are complex conditions that have their roots in biologic, psychologic and social issues. Predisposing factors to the development of EDs include being female, being perfectionistic and eager to please others, having difficulty communicating negative emotions such as anger, sadness and fear, having difficulty resolving conflict, and having low self esteem. The "typical" patient who presents with AN has been the perfect child, easy to parent. What precipitates the onset of the ED are issues related to adolescence—fears of maturation, struggles for independence and autonomy in middle childhood, and identity conflicts. As a "final common pathway," the eating disorder provides a way for the adolescent to distance herself from the stressors of adolescence. It also provides a way to control something in her life when she is feeling out of control. Perpetuating factors serve to maintain the eating disorder once the patterns of weight control are established. Biologic as well as psychological influences can be powerful reinforcers.

Starvation reinforces starvation; patients feel better, almost "high," when restricting. Patients who purge perceive themselves as overweight when normally hydrated. Psychologically, the eating disorder becomes a means of coping with fears about adolescence, and coping with family tur-

Abbreviations Used:

AN	anorexia nervosa
BMD	bone mineral density
BMI	body mass index
BN	bulimia nervosa
ED	eating disorder
ED NOS	eating disorder, not otherwise specified
IBW	ideal body weight
NG	naso-gastric

Table 1. DSM-IV Diagnostic Criteria

Anorexia Nervosa (307.1)

A. Refusal to maintain body weight at or above a minimally normal weight for age and height: weight loss leading to body weight less than 85% of that expected, or failure to make expected weight gain during period of growth.
B. Intense fear of gaining weight or becoming fat, event though underweight.
C. Disturbance in the way in which oneís body weight or shape is experienced, undue influence on body weight or shape on self evaluation.
D. In postmenarchal females, secondary amenorrhea (absence of three consecutive menstrual periods).
E. No other illness, medical or psychiatric to account for above.

Bulimia Nervosa (307.51)

A. Recurrent episodes of binge eating (eating a large amount of food in a short time during which there is a sense of lack of control over eating).
B. Recurrent inappropriate compensatory behavior in order to prevent weight gain: fasting, excessive exercise or self induced vomiting, misuse of laxatives, diuretics, enemas, etc.
C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for three months.
D. Self evaluation is unduly influenced by body shape and weight.

Table 2

Medical Complications of Anorexia Nervosa

Cardiovascular
cardiac muscle atrophy leading to ↓ wall size; ↓ heart size; bradycardia; hypotension; cardiac arrhythmias, mitral valve prolapse

Endocrine
anovulatory ammenorrhea (↓ FSH, ↓LH, ↓ estrogen); ↑ cortisol; ↑ growth hormone levels; ↓ somatomedin; normal prolactin; low/normal T4; normal TSH

GI
delayed gastric emptying; constipation; slight ↑ LFT's

Metabolic
hypercholesterolemia (early); hypocholesterolemia (late); carotenemia; low glucose, low insulin; decreased protein and zinc if malnutrition severe

Skeletal
osteoporosis; ↑ fracture risk (?irreversible)

Medical Complications in Bulimia Nervosa

Cardiovascular
EKG changes; peripheral edema; ipecac-induced cardiomyopathy; cardiac arrhythmias and arrest.

Dehydration, electrolyte abnormalities
Hypochloremic metabolic alkalosis; hypokalemia; hyponatremia; hypocalcemia; hypomagnesemia

Endocrine
Irregular menses; ↓ LH, FSH and estrogen; ↑ cortisol

Gastrointestinal
constipation; ↓ gastric emptying; GI bleeding; peptic ulcer; GER; pancreatitis; esophageal tear; malabsorption

Metabolic
↓ glucose?, ↓ insulin secretion and delayed insulin clearance

Skeletal
↓ BMD for age, ↑ fracture risk (?irreversible)

moil. The "power" derived from whether the patient eats or not, although not consciously sought, is hard to give up.

CLINICAL ASPECTS

Identifying a patient with an eating disorder can be difficult because symptoms may be exaggerations of normal teenage dietary fads. All adolescent patients should be questioned about exercise and eating habits on physical exam visits. Adolescent females should be questioned about their calcium and iron intake. Height and weight should be checked and BMI calculated. BMI is derived from weight (in kilograms) over height (in meters squared). It is highly correlated with body fat.[6] (Figure 1) In the absence of a BMI calculation, one can use a patient's weight and height to calculate their ideal body weight and then figure what percentage of ideal body weight they are (ideal body weight equals 100 pounds for 5 feet and 5 pounds for each inch above 5 feet for a female; 7 pounds for each inch for a male). Vital signs should be taken and particular attention should be paid to low pulse and temperature measurements, which may indicate some degree of poor nutrition.

Any weight loss in an adolescent who has not reached puberty is alerting. An adolescent who is significantly below ideal body weight should be asked about eating and exercise habits. Directly addressing the weight and nutritional issues is advisable. "I notice that your weight is quite low for your height—do you do anything significant to control your weight or eating?" Ask about intake for the past 24 hours and about exercise, focusing on the purpose of the exercise, i.e., how much is related to the need to burn calories. If a patient is dieting, ask if they have a weight loss goal in mind and tell her if it is not realistic. To get at body image issues and distortions, ask her if she is satisfied with her weight and shape. Self report questionnaires (Dying to be Thin and the Eating Disorders Inventory) can be useful in the evaluation of suspected eating disorder patients. The patient can privately answer questions with paper and pencil that she might have difficulty discussing.

Strenuous dieting in any girl of normal weight is a danger sign. Any teenager who restricts their food intake to 1000 calories a day or less or who tries to consume a zero fat diet is definitely at risk for an eating disorder. If a patient's sense of weight and shape is seriously distorted, an eating disorder may be present, even if significant weight loss has not occurred.

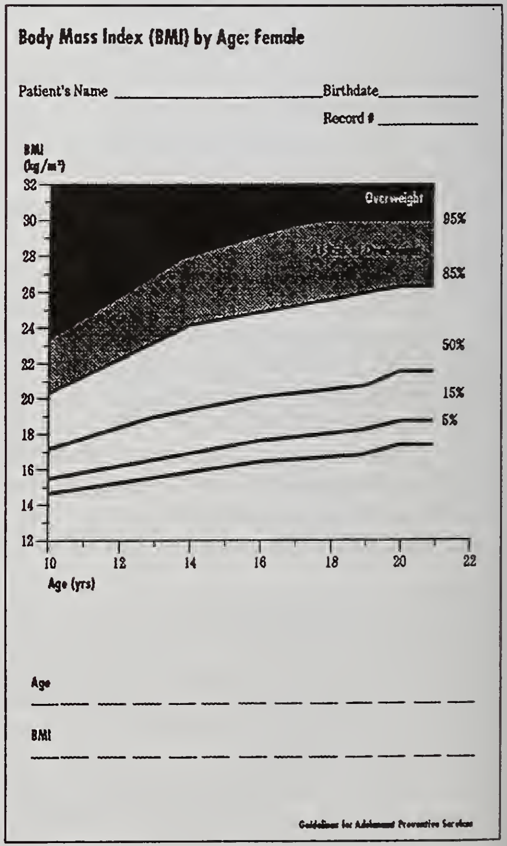

Figure 1.

Table 3. Role of the Primary Care Physician in Eating Disorders

- Early diagnosis; psychoeducation with patient and family
- Medical stabilization and refeeding
- Interval medical visits to monitor medical stability, labs and assess progress with nutritional rehabilitation
- Monitor (and limit) physical activity
- Convene a treatment team which will also (ideally) include a nutritionist, family and individual therapists
- Follow-up of amenorrhea, bone density, cardiac abnormalities
- Primary prevention efforts in schools, community

Strenuous dieting in any girl of normal weight is a danger sign.

In addition to obvious weight loss, history may reveal the avoidance of family meals or dramatic dietary changes (becoming vegetarian or vegan, avoiding all high calorie foods). Many adolescents who present with somewhat vague complaints of fatigue, weakness, general malaise or menstrual irregularity might have an eating disorder, as well.

CONFIRMING THE DIAGNOSIS

Once a physician has decided a patient may have an ED, he/she begins the diagnostic medical work-up (Tables 2 and 3). Clarifying, in terms that the patient can understand, the criteria for ED are and why she may be suffering from one is often very helpful. Patients and parents often mistakenly believe that a person has to be eating nothing to qualify for the diagnosis of AN or that BN involves purging, when it is really dependent on the binge phase of the behavior. A physician might point out the ways that an individual patient's signs and symptoms and physical characteristics reveal that her body is not healthy. The symptoms experienced and laboratory abnormalities found in a patient are related to the various habits she uses to control weight. A review of systems usually elicits a number of positive responses that reveal that all is not well. For example, loss of menstrual periods, cold hands and feet (acrocyanosis), dry and flaky skin (functional hypothyroidism), constipation and easy fatigability are usually evident. A low heart rate (less than or equal to 40) low core body temperature (less than or equal to 97°), and dehydration (as evidenced by high urine specific gravity), may also be mentioned to the patient in tying their findings to malnutrition. Many patients describe difficulty concentrating, making decisions, general irritability and obsessive thought about food, calories, and fat grams. Most patients have many physical symptoms of depression—such as early morning wakening—because malnutrition is usually accompanied by depressive symptoms. Patients with binge/purge ED may have different signs in addition of those of malnutrition. Bulimia is characterized by salivary gland enlargement from stimulation by binge eating and vomiting, dental enamel erosion from gastric acid and widely fluctuating body weight with intermittent edema.

TREATMENT ROLE OF THE PRIMARY CARE PROVIDER

Treatment of a patient with an ED begins with the assessment of weight loss described above. The physician should emphasize his/her role in guiding the patient to maintain health. (Table 3) It is also important to point out that EDs are usually chronic illnesses, with no "quick" "magic" cure; food is the medicine that is needed. The physician usually sets limits on weight loss: "I do not want you to lose any more weight." Limitation of physical activity is often also needed to conserve calories. A follow-up visit should be scheduled in a few days to a week to reassess weight, vital signs and nutritional progress. If a patient has a moderate or severe eating disorder, she should be referred to a counselor and a nutritionist experienced with ED. Ideally, the nutritionist, the physician, the counselor would be located in the same space to facilitate teamwork. The physician often serves as the case manager for the team's efforts.

HOSPITALIZATION

Indications for hospitalization in ED are outlined in Table 4. Hospitalization should never be seen as a punishment for the patient or family. Acknowledging that the cognitive distortion makes it impossible for the patient to alter her health-compromising behavior is helpful. It is important to outline for the patient and parent the purpose of the hospitalization as well as specific objectives (stabilize vital signs, begin refeeding, maintain vigi-

Table 4. Indications for Hospitalization

- Extreme weight loss (BMI < 15% for age, weight loss > 30% IBW)
- Unstable vital signs: bradycardia, low temperature, low blood pressure, orthostasis
- Dehydration, electrolyte disturbance unresponsive to outpatient treatment
- Symptoms of inadequate cerebral perfusion or altered mentation (syncope, severe dizziness, cognitive dysfunction).
- Depression, suicidal ideation
- Rapidly progressing weight loss (> 2 pounds in a week)
- Failure to progress in nutritional rehabilitation in outpatient treatment

lance for signs of refeeding syndrome, intensify psychotherapy). The goals of treatment are to help the patient achieve physical and emotional health. Medical and nutritional stabilization will likely be necessary before psychological therapy can be optimally effective for the patient with AN. For the patient with bulimia, the goal of a hospitalization is to sustain a normal intake without restricting, bingeing or purging. Utilization of a behavioral refeeding contract often helps to standardize the treatment for nursing personnel. A patient is required to eat all food in a minimal amount of time and be supervised in the bathroom. Her ability to undertake increasing levels of physical activity is contingent upon regular small incremental weight gain. NG tube feedings are infrequently required but may be needed when the patient is unable to tolerate enough food orally to gain adequate weight. Extremely malnourished patients who are hospitalized for restrictive ED are at risk for "refeeding" syndrome[7] if they are fed too many calories to quickly. These hypometabolic adolescents may require as few as 800 to 1,000 kilocalories for the first few days. As resting energy expenditure increases with refeeding, the caloric intake may be increased to reflect metabolic recovery. Refeeding syndrome is the term used to describe those metabolic complications that are seen with rapid repletion of calories, causing fluid shifts and alterations in serum potassium, magnesium, glucose and minerals. Many of the symptoms of refeeding syndrome are the result of severe hyposphatemia (altered cardiac function, arrhythmia, heart failure, hepatic dysfunction, confusion, coma and seizures).

Table 5. Resources for Adolescents and their Families

IN RHODE ISLAND
Medical evaluation and treatment:
Adolescent Health Clinic
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Comprehensive inpatient and outpatient medical evaluations, including nutritional assessment and eating disorder questionnaires. On-going eating disorder and nutritionist care. For new patients 18 years and under.

Psychological assessment and care:
Hasbro Partial Hospital Program
Dr. Thomas Roesler
(401) 444-5617

Outpatient partial hospital care for youngsters with medical/psychological problems, including eating disorders. For patients 18 years and younger.

Butler Hospital Eating Disorders Program:
Dr. Leigh Anne Hohlstein
(401) 455-6228

Outpatient and partial hospital psychiatric care for older adolescents and young adults.

Anorexia and Bulimia Association of RI:
Dr. Steven Emmett
(401) 861-2335

NATIONAL SUPPORT GROUPS:
National Resources
American Anorexia/Bulimia Association
418 East 76th Street
New York, NY 10021
(212) 734-1114
http://members.aol.com/amanbu

Academy for Eating Disorders
6728 Old McLean Village Drive
McLean, VA 22101-3906
(703) 556-9222
E mail:aed@degnon.org
www.acadeatdis.org

E.D.A.P. Eating Disorders Awareness & Prevention
255 Alhambra Circle, #321
Coral Gables, Fl 33134
(305) 444-3731

At the Hasbro Children's Hospital, a "critical path" for the management of inpatients with ED has been developed and used for the past 15 months. It includes a seven day plan to stabilize and refeed a malnourished patient. Discharge criteria for inpatient care include achievement of an adequate caloric intake, gradual weight gain and correction of some of the patient's cognitive distortions (usually when weight approaches 10% ideal body weight). The path ensures a standard of care and has been favorably accepted by payors as well as staff. To date approximately 50 patients per year have been managed using the care "map."

POTENTIAL LONG TERM SEQUELAE

Secondary amenorrhea and osteoporosis occur in more than 50% of female patients with AN. Bone mineral density (BMD) deficiencies persist in 85% of partially recovered eating disorder patients who have resumed menses and attained weights within 10% of ideal body weight. Evidence of BMD deficiency has been detected within one year of the onset of AN and BN in adolescents.[8] Although most studies have been on female patients, there is evidence that males with AN may also develop osteoporosis. Multiple factors increase an adolescentís risk for osteoporosis or osteopenia: decreased body weight and fat content, elevated cortisol, inadequate vitamin D and calcium intake, amenorrhea and hypoestrogenemia. There is both decreased bone formation and increased bone resorbtion in the osteoporosis of AN. Treatment includes weight normalization and supplementation with calcium and vitamin D; it may also include exercise limitation when the patient is at extremely low weight. Unlike post-menopausal osteoporosis, estrogen replacement therapy does not prevent or correct the osteoporosis that occurs in AN. Although resumption of menses usually occurs when the patient is within 10% of IBW and has a normalized serum estradiol level, recovery from AN is not associated with normalization of BMD. Among the most striking physical consequences of AN in adolescents (and adults) are the structural abnormalities in the brain. Controlled studies in adults and teenagers have shown ventricular and cortical sulcal enlargement. It is not known whether these changes are entirely reversible and they may be related to reported neuropsychological effects in some patients (impaired attention and concentration, and psychomotor slowing).[1]

OUTCOME STUDIES

To date no studies have evaluated the outcome of a group of exclusively adolescent patients with BN. A number of studies have evaluated the short and long-term outcome of patients with AN. Most of these studies have combined patients who are adolescents with adult patients. Studies of AN have generally demonstrated that approximately 50% of patients have a good outcome, 25 % have an intermediate outcome and 25% do poorly. Adolescents with AN generally have a better prognosis than adults. Poorer outcomes have been associated with later onset and longer duration of illness, more disturbed premorbid personality and presence of bulimic behaviors. Early age of onset has actually been proposed as a predictor of good outcome since younger patients generally present with less severely entrenched psychiatric difficulties.[1] Three groups have published outcome data on a total of 153 adolescent patients treated for AN.[9-11] Twenty percent of the adolescents were treated solely as outpatients whereas 80% required a combined inpatient and outpatient approach. All patients were evaluated with the Global Assessment Scale of Garfinkel (evaluates outcome in terms of weight, menstruation, eating habits, social adjustment and education and employment). When the findings of the three studies are combined, 75% of patients were categorized as having an excellent or much improved outcome at a mean of four years of follow up, whereas 25% remained symptomatic. Mean weight at follow-up was 94% of IBW; 79% had resumed menses; 81% reported no vomiting and most reported good social functioning, educational achievement and employment. The finding that most adolescents with ED do well with treatment (unlike many adults) supports early aggressive treatment in adolescents. However, we also need to work on primary prevention, encouraging all children and adolescents to develop healthy attitudes about weight, body image and self esteem and to ameliorate the sociocultural influences that promote ED.

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Depressive Disorders in Adolescents:
A Review for Adolescent Health Care Practitioners

Alessandra N. Kazura, MD

Abbreviations Used:	
BDI	Beck Depression Inventory
ECT	electroconvulsive therapy
MAOI	monoamine oxidase inhibitor
MDD	Major Depressive Disorder
SSRI	selective serotonin reuptake inhibitor
TCA	tricyclic antidepressant

Although Major Depression and other depressive disorders are common in adolescence, patients are frequently undiagnosed and/or undertreated. Given the significant morbidity, and even mortality associated with mood disorders, adolescent health care practitioners need to be able to identify and refer these patients for appropriate mental health services.

DISORDERS WITH DEPRESSED MOOD

A number of conditions may present with prominent depressed mood. Major depression requires the presence for two or more weeks of a cluster of symptoms that include depressed mood and/or anhedonia. These symptoms are (1) depressed or irritable mood; (2) loss of interest or pleasure; (3) weight loss or gain, or appetite loss or gain; (4) insomnia or hypersomnia; (5) psychomotor agitation or retardation; (6) fatigue or energy loss; (7) feelings of worthlessness or guilt; (8) diminished concentration or indecisiveness; and (9) recurrent thoughts of death, with or without suicidal ideation or attempts.[2] Bipolar disorders must be ruled out, as well as mood disturbance due to a substance, general medical condition, or bereavement.[2]

Even in the presence of an associated stressful event, Adjustment Disorders are not diagnosed if the full criteria for a Major Depressive Episode are met. When multiple symptoms are present after the death of a loved one, Major Depressive Disorder (MDD) is distinguished from Bereavement by the continuation of these symptoms for more than two months or by the presence of significant functional impairment, suicidal ideation, psychotic symptoms, psychomotor retardation, and preoccupation with worthlessness. Severity of symptoms, duration, and associated impairment distinguish normal sadness from a disorder.[2]

EPIDEMIOLOGY OF DEPRESSION

In 1995, 42% of Rhode Island 7th-12th grade students responded "yes" to the question: "In the past 6 months, have you had the feeling that life was not worth living?"[10] While this does not tell us how many were depressed, it does serve as a proxy marker for feelings of hopelessness that may be part of the presentation of depression.

In adolescents, lifetime prevalence rates of Major Depressive Disorder are on the order of 15-20%.[3] Females experience depression at about twice the rate of males, reversing the preadolescent male predominance of depression.[7] Cohort studies indicate that the age of onset of depression has become earlier in successive cohorts.[6, 12]

Table 1. Evaluation of Depression

Interviews with adolescent, parents, and other key informants
- Onset and pattern of mood symptoms
- Current and baseline level of function
- Exposure to stressful life events
- Suicidal ideation
- Substance use
- Strengths/weaknesses in social support system
- Medical history and review of symptoms
- Current medications
- Psychiatric treatment history
- Mental status examination

Physical Examination
Laboratory Tests
- As determined by evaluation and/or need for pre-medication screening

Rating Scales (examples)
- Beck Depression Inventory
- Global Assessment of Functioning

RISK FACTORS

Major Depressive Disorder is associated with multiple risk factors. Genetic vulnerability is fairly well established by many twin, adoption, and family aggregation studies.[1] Other biological vulnerabilities may include abnormalities in affect regulation which is likely transmitted by dysfunctional neurotransmitter and neuroendocrine systems.

Personal psychological factors may include conflict over dependency needs, high degree of self-criticism, low self-esteem, and pattern of negative attributions. Family conflict, rejection, or indifference are also associated with depression. Exposure to stressful life events is more common in depressed than nondepressed adolescents, particularly the death of a parent or other loved one, but also divorce of parents, moves, and traumatic experiences.

Table 2. Antidepressants ^{a,b}

Name	Typical Start Doses (mg)	Typical Therapeutic Ranges (mg)	Side Effects
Citalopram	10 - 20	10 - 60	SSRIs: **common:** nausea, diarrhea, headache, anxiety, insomnia, restlessness, dry mouth, sedation, orthostatic hypotenxion, tremor, sweating, apathy, decreased sexual drive **less common:** weight loss, hypromania, weight gain serious: urticaria, seizures, heatstroke, serotonin syndrome
Fluoxetine	10	2.5 - 60	
Fluvoxamine	50	50 - 200	
Paroxetine	10	5 - 60	
Sertraline	25	25 - 200	
Buproprion	75	75 – 450 div	**common:** nervousness, dry mouth, constipation, headache, weight loss, nausea, dizziness, sweating occasional: tics, myoclonus, insomnia, rash, perioral swelling **less common:** vomiting, seizures, hypomania
Venlafaxine	37.5	75 – 200 div	**common:** anxiety, nausea, insomnia, decreased appetite, weight loss occasional: yawning, blurred vision, orthostatic hypotension, constipation, sweating, lack of energy, trouble with sexual functioning **less common:** hypertension, seizures
Nefazodone	25	50 - 400 div	**common:** sedation, dry mouth, dizziness, headache, blurred vision, nausea, more frequent erections **less common:** tachycardia, syncope, vomitory, neutropenia, priapism, increased sexual drive
Trazodone	50	25 – 300	
Mirtazepine	5	15 – 45	**common:** sedation, dry mouth, increased appetite, weight gain, constipation **less common:** asthenia, flu-like syndrome, back pain, edema, dizziness, abnormal dreams, tremors, confusion, urinary frequency, dyspnea
Amitriptyline	10-25	25 - 300	TCAs: **common:** dry mouth, constipation, orthostatic hypotension, weight gain, loss of appetite, weight loss, sedation, irritability **occasional:** nightmares, shuttering, sun sensitivity, gynecomastia, decreased sexual drive **less common:** hypotension, hypertension, nausea, blurred vision, tics, activation/hypomania **rare:** seizures, dysrrhythmia, urinary reteation, hallucinations, confusion
Clomipramine	25	50 - 200 div	
Doxepin	25	50 - 300	
Imipramine	10-25	20 - 450 div	
Desipramine	10-25	25 - 300 div	
Nortriptyline	10-25	25 - 200 div	
Protriptyline	5	10 - 60 div	
Trimipramine	25	25 - 300	
Amoxapine	25	25 – 300	dizziness, sedation, anticholinergic effects, seizures, akathisia, dytonia, dyskinesias, agranulocytosis, hypotension, tachycardia, dysrhythmia, gynecomastia, rashes
Maprotiline	25	50 –225	neutropenia, tachycardia, hypotension, hypertension, sedation, vertigo, tremor, anxiety. seizures, hallucinations, hypomania, myo clonus, weight gain, nausea, vomiting, dry mouth, constipation, urinary retention, LFT elevations, blurred vision
Isocarboxazid	5	10-30 div	MAOIs: tyramine associated pressor effect (requires strict dietary protocol), hypotension, hypertension, dizziness, insomnia, impotence, edema, weight gain, LFT, elevation, tremors, twitching, psychotic symptoms, serotin syndrome, hypomania
Moclobemide	100	100 - 600 div	
Phenelzine	15	15 - 45 div	
Tranylcypromine	10	10 - 30 div	

a.) Adapted from Werry & Aman, 1999; Dulcan, 1999; Rosenberg et al., 1994; Micromedex ®

b.) Many have very limited use in adolescents (see text).

EVALUATION

A comprehensive evaluation is critical for establishing the diagnosis and for tailoring treatment choices. This is described in detail in *Practice Parameters for the Assessment and Treatment of Children and Adolescents with Depressive Disorders.*[1]

The initial evaluation should include details of the mood disturbance, including onset, severity, and fluctuation. In addition to sad or depressed mood, adolescents should be asked about feelings of anxiety, which commonly precede depression and feelings of low self-worth. Parents are relatively poor reporters of their children's experience of feelings, but should be asked about observations of irritability, loss of pleasure or interest in usual activities, social isolation, and deterioration in academic performance.

Neurovegetative symptoms include changes in sleep pattern (both increases and decreases in duration of sleep) and in appetite and weight. Difficulties in concentrating and decision-making are often reported; they may be reflected in deterioration of grades.

Suicidal ideation may be present, and it will require careful evaluation for acuity of dangerousness. Risk factors that are associated with suicide include a past history of suicidal behavior, impulsivity, hopelessness, availability of lethal means, a family history of suicide, family conflicts, and exposure to abuse or other violence.

Psychotic symptoms, such as delusions or hallucinations, may be subtle, but predict greater severity of depression, higher risk of bipolar disorder, and resistance to standard therapies. Systematic use of a thorough mental status examination will help with the detection of these serious symptoms.

Screening for comorbid psychiatric conditions is critical. Epidemiological studies report overall rates of comorbid psychiatric conditions to be 40-70%, with the most common disorders being Dysthymic Disorder, anxiety disorders, disruptive disorders, and substance abuse/dependence disorders; 20-50% of adolescents with MDD will have two or more comorbid diagnoses.[1]

A medical history, with a review of symptoms and a physical examination, are part of the standard initial evaluation of depression. Medical conditions that may cause or contribute to depressive symptoms include infections such as mononucleosis, hepatitis and human immunodeficiency virus; neurological disorders such as epilepsy and postconcussion syndrome; endocrine disorders such as hypothyroidism, diabetes, and Cushing's disease; nutritional deficiencies; autoimmune disorders such as lupus; and reactions to medications including contraceptive agents, steroids, anticonvulsants and benodiazepines. Since no laboratory test can confirm the diagnosis of MDD, the request for blood work or other studies should be guided by concerns raised during the medical examination and/or by consideration of baseline screening assessments that may be needed for biological therapies. Pregnancy screening should be considered for females.

Rating scales can assist in treatment monitoring. The Beck Depression Inventory has the advantages of widespread clinical use and ease of administration.[8] The BDI helps with the documentation and tracking of the severity of the depression, but because its items are nonspecific, it does not establish the diagnosis of depression. Global functioning scales (e.g., the Global Assessment of Functioning) are useful in documenting the degree of impairment at baseline and during the course of treatment. [2] (Table 1)

TREATMENT: BIOLOGICAL

Over the last 10 years, there has been an explosion in the number of antidepressants, although relatively few have been tested with rigorous randomized, placebo-controlled trials in adolescents or children. The data suggest that many adolescents will experience benefits from antidepressants. Also, medications have clinical uses in addition to depression. For example, some may be beneficial for symptoms of Attention Deficit Hyperactivity Disorder, Obsessive Compulsive Disorder, and Panic Disorder. These properties can be used to advantage in the treatment of patients with comorbid conditions.

Initial positive response to antidepressants may take two weeks or more at therapeutic doses. One of the more common clinical mistakes is to declare a medication trial a failure before adequate doses and duration are achieved. Decisions about how long to continue medication have been extrapolated from continuation and maintenance studies in adults and from accumulated clinical practice with adolescents. In general, treatment should be continued for 16-20 weeks after full remission of the depressive disorder; patients with recurrent depression or severe depression may need long-term maintenance.

Selective serotonin reuptake inhibitors (SSRIs) are the most widely prescribed class of antidepressants in adolescents, which is not surprising given their safety and side effect profiles compared to the older tricyclic and MAO inhibitor antidepressants. (Table 2) No single agent has proven superior to others in this age group, and failure to respond to one SSRI does not predict failure to respond to the others. With the exception of sertraline and citalopram, SSRIs can inhibit the P-450 isoenzymes; therefore, these medications can have clinically significant interactions with other drugs metabolized by these enzymes.

Serotonin syndrome is a relatively uncommon, but serious, medical complication of SSRI use, and it should be considered when patients on these agents present with confusion, agitation, chills, muscle twitching, diaphoresis, fever, nausea, vomiting, or diarrhea. Early recognition with discontinuation of the serotonergic agent and supportive medical care are often adequate for resolution.

Older antidepressants include tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs). The limited studies of TCAs and MAOIs in adolescents do not demonstrate superiority over placebo, unlike the experience in adults. Side effects tend to be high in children and adolescents, and overdose is associated with a significant risk of death, so these agents are not usually considered first line treatments.

Numerous other antidepressants may be beneficial for a given individual. (Table 3). Of particular note are bupropion, mirtazepine, venlafaxine, and nefazodone. These medications offer options to serotonergic agents. No biological test is available to predict probability of success with any of these medications. Side effect profile and efficacy for comorbid conditions will help to determine initial and subsequent antidepressant choices.

Herbal therapy, specifically St. John's Wort (Hypericum), has been recently popularized. Evidence for efficacy in adults is limited. One caveat: combining St. John's Wort with other biological therapies may be problematic. For example, simultaneous use of SSRIs with St. John's Wort may increase the risk for serotonin syndrome.

Although rarely used in adolescents, Electroconvulsive Therapy (ECT) deserves mention as a treatment for depression that is resistant to other therapies. Efficacy data are limited, but suggest that treatment response rates range from 50-65%, similar to rates seen in adults. Reported side effects to ECT in adolescents have included confusion, agitation, subjective memory loss, headaches, vomiting, and manic switches.[9]

Numerous augmentation and maintenance strategies are in clinical use, but are beyond the scope of this paper.

TREATMENT: PSYCHOSOCIAL

Psychotherapeutic modalities may be the initial treatment for MDD, especially for first episodes and mild to moderate episodes.[1] Combinations of psychosocial and biological treatments are common in clinical practice. In adults, combined treatments have resulted in better response rates than single modality therapies. Nonspecific issues to incorporate include support, education about the nature and course of the illness, and adherence to treatment. A variety of theoretically driven therapies are currently in use. Cognitive behavioral therapy is based on observations that depressed patients have negative attributions about themselves, their world, and their future. Therapeutic techniques include teaching patients to challenge these negative attributions, and teaching them stress management and problem-solving skills. Interpersonal therapy is based on the association of mood symptoms with dysfunctional patterns of interpersonal relationships, and addresses problems with conflict management, interpersonal roles, and grief. Self esteem issues, clarification of feelings, development of insight about dysfunctional behaviors, and coping with intrapsychic and external conflicts may be addressed in psychodynamically-based therapies.[1]

Group therapies may derive content and process elements from some of the same theoretical sources as listed above, but they all share the format of utilization of relationships between peers as a force for change.

Family therapy is a reasonable primary or adjunctive treatment. Conflict between family members, inadequacy of support, behavioral control problems, and poor communication are just some of the areas that may be targets of the therapy. Screening for other family members for depression and other disorders is especially important since rates of parental mental illness are quite high in adolescents with depression, and parental dysfunction may be a serious barrier to the depressed adolescent's recovery.

Depressive disorders are common in adolescents...

Factors that determine treatment selection include specific symptom clusters, comorbid psychiatric and/or medical conditions, response of family members to specific biological agents, side effect profiles that are most acceptable to patients, severity and duration of the current episode, history of recurrent depressive symptoms, psychotic features, prior patient experience with a specific treatment, and practitioner experience with specific treatments.

Hospitalization is indicated when outpatient therapy has failed, when suicidal and/or other aggressive impulses are a concern, or when comorbid conditions such as substance abuse present a barrier to treatment. Factors such as a family's ability to closely supervise and maintain patient safety and to support treatment adherence should be factored into the decision. Depending on resources and patient circumstances, options include intensifying outpatient care with partial hospital programs, therapeutic school settings, and home-based services.

COURSE AND PROGNOSIS

In adults, earlier initiation of treatment has been associated with shorter duration of the depression. While this has not been studied in adolescents, clinical wisdom supports assertive identification and treatment of symptoms as soon as possible. Prevention studies using cognitive therapies in children and adolescents at high risk for development of major depression due to subsyndromal symptoms or family risk factors have reported delays in onset of depressive episodes.[4]

The typical mean duration for Major Depression and Dysthymia is 7-9 months and 4 years, respectively.[3] About 70% of adolescents with depression will experience a recurrence within 5 years and 20-40% will go on to be diagnosed with Bipolar Disorder.[3] The presence of family conflict is a major risk factor for recurrence.[3]

ROLE OF THE PRIMARY CARE PROVIDER

Primary care providers have important roles in working with depressed adolescents. Routine screening for signs and symptoms of depression will contribute to increased identification and referral to mental health clinicians. Comprehensive medical evaluations will assist in identifying contributing medical conditions.

Unfortunately, mental disorders continue to be associated with social stigma. Adolescents and their parents may need coaching from the primary care physician before they will accept referral and treatment. Education about the nature of depression, and preparation for what to expect with the mental health evaluation may be helpful. In addition, the primary care physician can support treatment adherence and can assist with the monitoring of biological therapies.

Additional sources of information and support for patients and their families may be obtained through well-established organizations such as the National Alliance for the Mentally Ill (1-800-950-NAMI; www.nami.org) and the National Depressive and Manic Depressive Association (1-800-826-3632; www.ndmda.org), both of which have Rhode Island chapters. A useful source of medication information for adolescents and their parents is *Helping Parents, Youth and Teachers Understand Medications for Behavioral and Emotional Problems: A Resource Book of Medication Information Handouts.*

CONCLUSION

Depressive disorders are common in adolescents and may be associated with serious consequences including suicide, academic and social impairment, and development of additional psychiatric disorders. Adolescent health care providers can have a beneficial impact on the course of these disorders by routinely screening for mood symptoms, educating patients and families, and referring for comprehensive evaluation and treatment.

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Cardiac Causes of Syncope in the Adolescent

Lloyd R. Feit, MD

Syncope is defined as the sudden loss of consciousness and postural tone resulting from an abrupt diffuse, transient cerebral malfunction followed by spontaneous recovery. It is due to a sudden reduction in delivery of a vital substrate to the brain, usually oxygen. In some patients, loss of consciousness may be preceded by a state of pre-syncope, which may be defined as a prodrome of distinctive premonitory symptoms including dizziness, lightheadedness, pallor, diaphoresis and palpitations. Most studies suggest that roughly 50% of adolescents will have experienced at least one episode of syncope.[1] In one retrospective study, syncope accounted for almost 1% of pediatric emergency department visits over a one-year period.[2]

The causes may be categorized into autonomic, cardiac and noncardiac. (Table 1) Over 75% of referrals for syncope in teenagers are for benign vasovagal syncope.[3,4]

CARDIAC CAUSES OF SYNCOPE

In the teen presenting with syncope, the primary concern is whether the event is a marker for sudden death. This is especially true after the rare death of a sports figure. The prevalence of athletic field deaths in high school is thought to be 1 in 100,000 to 1 in 300,000.[5] Most sudden death in young athletes can be linked to cardiac conditions, usually not previously known. [6] Hypertrophic cardiomyopathy and anomalies of the coronary artery distribution, account for over half of the identifiable cardiac causes for sudden death in athletes.[6] Although not always associated with a risk of sudden death, other cardiac causes of syncope (including some arrhythmias) may be amenable to corrective therapy.

Abbreviations Used:

HOCM	hypertrophic obstructive cardiomyopathy
LQTS	long QT syndrome
SVT	supraventricular tachycardia
VT	ventricular tachycardia
WPW	Wolff-Parkinson-White

OBSTRUCTIVE LESIONS

Obstruction to ventricular outflow, or (more rarely) ventricular inflow can be associated with syncope. This has been best described in patients with severe aortic stenosis or hypertrophic obstructive cardiomyopathy (HOCM), but may also be seen in patients with pulmonic stenosis, mitral stenosis, atrial myxoma, cardiac tamponade or primary pulmonary hypertension. Unrecognized HOCM is the most commonly identified cause of sudden death in young athletes. Arrhythmia plays an especially important role in HOCM, since the risk of sudden death is increased in those pa-

Table 1: Etiology of syncope

Autonomic

- Vasovagal (most common cause of syncope in children)
- Excessive vagal tone (athletes, adolescents)
- Reflex
  - Situational (cough, micturition, hair grooming, etc.)
  - Pallid breath-holding
- Orthostatic (dehydration, blood loss)

Cardiac

- Obstructive lesions (aortic stenosis, hypertrophic obstructive cardiomyopathy, primary pulmonary hypertension)
- Arrhythmia (SVT, VT, heart block)
- Hypercyanosis ("Tet spells")
- Miscellaneous (pump dysfunction, myocardial infarction, anomalous coronary anatomy)

Non-cardiac

- Neurologic (seizures, migraine)
- Metabolic (hypoglycemia)
- Hyperventilation
- Hysterical (audience, complete absence of trauma)
- Vascular (cervical anomalies, vertebro-basilar insufficiency)

All patients who have syncope during exertion should be evaluated by a cardiologist.

tients who had previous syncopal episodes or documented ventricular tachyarrhythmias, and is not related to the degree of outflow obstruction.

Syncope noted with specific positional changes may suggest a pedunculated (left) atrial myxoma intermittently causing obstruction to ventricular inflow Recurrent syncope in an otherwise healthy adolescent female may suggest the diagnosis of primary pulmonary hypertension, or recurrent pulmonary embolism associated with oral contraceptive use.

ARRHYTHMIAS

Arrhythmias are a less frequent cause of syncope in children than in adults. One logical approach distinguishes arrhythmias as a cause for syncope based on the presence or absence of structural heart disease. The mechanism causing syncope common to all arrhythmias is diminution of cardiac output to the point where cerebral perfusion cannot be maintained.

STRUCTURALLY NORMAL HEARTS

Probably the most common arrhythmia causing syncope in patients with a structurally normal heart is supraventricular tachycardia (SVT), especially in the context of Wolff-Parkinson-White (WPW) syndrome. Usually, the patient with SVT complains of palpitations long before cardiac output is compromised sufficiently to diminish cerebral perfusion. In the setting of atrial flutter or fibrillation, rapid conduction from atria to ventricles may permit ventricular rates up to 300-400 beats per minute, and sharp and sudden diminution of cardiac output.

Ventricular tachycardia (VT) may also cause syncope in patients with a normal heart. The causes are uncommon, and include infectious myocarditis, dilated cardiomyopathy, drug induced (cocaine, amphetamines, etc.), and congenital and acquired forms of the long QT syndrome (LQTS). Patients with LQTS present with syncope or "seizures" due to paroxysmal episodes of rapid VT. The diagnosis is made by documenting prolongation of the corrected QT interval (QTc ≥ 0.45 sec) on the resting ECG. Prolongation of the QTc interval results in concomitant prolongation of the refractory (vulnerable) period of the ventricular myocardium, placing it at risk for *torsades de pointes*, an especially malignant form of VT. The congenital type is comprised of two forms, distinguished by their pattern of inheritance. One follows an autosomal dominant pattern, (Romano-Ward syndrome), the other is inherited in an autosomal recessive pattern (Jervell Lange-Nielsen syndrome) and is often associated with congenital nerve deafness. In the acquired form, the QTc interval is prolonged due to electrolyte imbalance (hypokalemia, hypocalcemia, hypomagnesemia), CNS abnormalities (increased intracranial pressure) or medication use/overdose (eg tricyclic antidepressants, antiarrhythmic agents, antihistamines). Other medications that can prolong the QTc interval are nonsedating antihistamines, and cisapride . Patients on these medications chronically should have periodic evaluation of their ECG, with particular attention to the QTc interval. Other drugs that may induce ventricular arrhythmias include cocaine and anabolic steroids.

Excessively slow rhythms (sinus bradycardia and high grade AV block) can occur in adolescents and may infrequently present with loss of consciousness. Lyme carditis has been noted to cause heart block that is generally reversible, but may cause syncope.

STRUCTURAL HEART DISEASE &/ OR PRIOR CARDIAC SURGERY

Syncope in a child with structural heart disease including those surgically repaired, must be considered due to an arrhythmia until proven otherwise.

Any patient who has had a ventricular incision (ventriculotomy) as part of their repair is at risk for VT. This is particularly true if there is a residual hemodynamic load on the ventricle, as in repaired tetralogy of Fallot. Similarly, patients who have undergone any intra-atrial surgery are particularly prone to SVT as well as atrial flutter or fibrillation. This is a common finding in patients who have undergone atrial switch repair (Mustard or Senning procedure) for transposition of the great arteries. These patients pose a special therapeutic challenge, as they are also prone to significant bradycardia (tachy-brady syndrome).

Severe bradycardia and heart block may be seen in any patient who has had surgical manipulation in the area of the sinoatrial or atrioventricular nodes, especially those with AV canal defects, large posterior VSD, or severe tunnel sub-aortic stenosis. Patients without apparent heart disease with heart block may in fact have congenitally corrected transposition of the great arteries (L-TGA), and this should be ruled out echocardiographically in an otherwise normal child with syncope and heart block.

HYPERCYANOSIS

Patients with unrepaired cyanotic congenital heart disease are at risk for syncope when cyanosis becomes severe. This has been particularly well described in the patient with tetralogy of Fallot where syncope occurs due to profound cerebral hypoxemia.

MISCELLANEOUS

Rare abnormalities of coronary anatomy (Figure 1) may predispose to arrhythmia, myocardial infarction and syncope and are the second most common diagnosis identified on post mortem exam.[6] They should be strongly considered whenever syncope occurs with exercise.

Rare cardiac etiologies may result in syncope with the common pathophysiology of diminished cardiac output and cerebral hypoperfusion. Severe myocardial pump dysfunction may be observed in the setting of infection (myocarditis), myocardial infarction (Kawasaki syndrome, cocaine or steroid abuse) or may be idiopathic (dilated cardiomyopathy).

Table 2: Who gets referred?

- Exertional syncope
- Syncope associated with chest pain or palpitations
- Any cardiac abnormality on physical exam or ECG
- Family history of sudden death
- Family history of seizures
- Any neurologic abnormality or residua
- Unexplainable or atypical episodes
- Recurrent episodes

DIAGNOSTIC EVALUATION

HISTORY

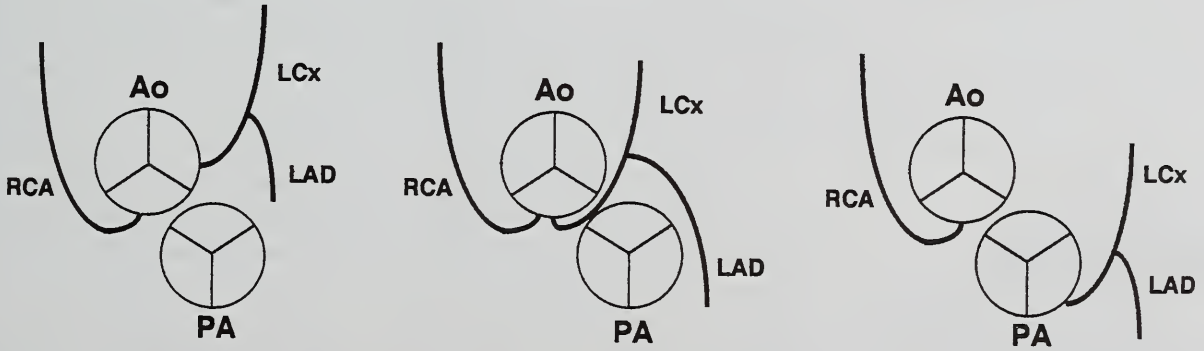
A complete and thorough history will in the majority of patients allow a diagnosis of vasovagal syncope or guide the clinician in the choice of invasive or noninvasive testing. A detailed description of the scenario in which syncope occurred , including activity and position of the patient immediately prior to loss of consciousness, presence of presyncopal symptoms, and the location of the event, all offer clinical clues to the diagnosis. Presyncopal symptoms typical of vasovagal syncope may be well described only by an eyewitness. Vasovagal syncope in the recumbent position is exceedingly rare.

Other scenarios of situational syncope include stretching, micturition, hair grooming, cough, and swallow. Emotional stress or underlying illness may intensify the tendency to faint, especially in those patients with a known history of vasovagal syncope. It may also be exacerbated by hot, humid, or crowded settings. Recurrent syncope in a regular location or social setting should suggest the possibility of hysterical syncope, especially if it does not result in noticeable trauma. Syncope that occurs after a sudden startle reaction suggests the long QT syndrome.

Vasovagal syncope typically lasts less than 1-2 minutes unless the patient attempts to stand up prematurely, and complete recovery occurs usually within 30-60 minutes. Longer syncopal episodes or those followed by an extended period of drowsiness suggest a seizure disorder.

Syncope during exercise or physical activity should always raise the question of a cardiac abnormality, suggesting inability to maintain cardiac output to meet increased demands. Although rare, typical anginal symptoms may suggest the diagnosis of a coronary artery anomaly and palpitations prior to the syncopal event may

Figure 1: Coronary artery anomalies causing syncope



Normal coronary anatomy

Anomalous LCA from Right sinus of Valsalva

Anomalous LCA from Pulmonary artery

Abbreviations: Ao: aorta, PA: pulmonary artery, LCA: left coronary artery, LAD: left anterior descending branch, LCx: left circumflex branch, RCA: right coronary artery.

suggest a tachyarrhythmia.

Antihypertensive drugs may induce syncope via excessive vasodilation and hypotension. Antiarrhythmic medications in class IA (esp. quinidine and procainamide) and class III (amiodarone and sotalol) may predispose to the *torsades de pointes* type of ventricular tachycardia by prolonging the QTc interval. The propensity to this arrhythmia may be exacerbated by diuretics or other medications that induce important electrolyte abnormalities such as hypokalemia and hypocalcemia. Antihistamines such as astemizole have also been implicated in the occurrence of ventricular tachycardia resulting in syncope and sudden death, though primarily associated with concomitant use of erythromycin or ketoconazole which decrease its metabolism. Illicit drug use may also cause syncope, possibly associated with arrhythmia or myocardial infarction (steroids, cocaine) or altered sensorium (marijuana, alcohol).

There is frequently a familial tendency for typical vasovagal syncope, which may be as high as 90%.[7] Family history of syncope, seizures or unexplained sudden death may also identify those at risk for long QT syndrome, hypertrophic obstructive cardiomyopathy, WPW or arrhythmogenic right ventricular dysplasia.

A past medical history of cardiac disease, especially if surgically repaired, should place cardiac syncope high on the differential.

PHYSICAL EXAMINATION

All patients with syncope should have a complete physical examination, including a thorough cardiac and neurologic assessment. Palpation of the precordium may suggest pulmonary hypertension (parasternal right ventricular heave), left ventricular hypertrophy seen with left ventricular outflow obstruction (left, inferior displacement of the apical impulse), or left ventricular dysfunction (absent apical impulse). Auscultation should identify murmurs of aortic and pulmonic stenosis and HOCM, the gallop rhythm of any generalized cardiomyopathy, or the apical diastolic murmur of mitral stenosis. Baseline and orthostatic vital signs should be carefully recorded. In an adolescent, after 5-10 minutes standing, a systolic blood pressure of less than 80 mmHg or a decrease of > 30 mmHg suggests orthostatic physiology

DIAGNOSTIC CARDIACTESTING

Every patient evaluated for loss of consciousness should receive a baseline ECG, useful in making the diagnoses of Wolff-Parkinson-White syndrome, long QT syndrome, heart block, myocardial ischemia and/or infarction, or other forms of congenital heart disease. If an arrhythmia is suspect, more long term ECG monitoring is indicated such as 24-hour Holter monitoring, or event recorders for intermittent symptoms.

If structural cardiac disease is suspected , an echocardiogram is indicated. This is especially important in the setting of familial cardiomyopathies, where the cardiac exam may be normal. Those patients with a history of exercise induced syncope may benefit from a physician supervised exercise stress test. Cardiac catheterization maybe helpful in those with suspected coronary abnormalities. Electrophysiologic studies are only indicated if there is a strong suspicion of potentially lethal arrhythmia or a history of surgically repaired CHD.

Tilt table testing is generally reserved for use in the patient with frequent or atypical syncopal episodes in whom the diagnosis of vasovagal syncope cannot be definitively made. Reported sensitivity for induction of syncope using tilt testing has ranged from 30% to 80%, probably related in part to differing protocols and endpoints. Tilt testing identifies the patient with a tendency to vasovagal syncope and may be variably present in a patient, giving rise to a high rate of false positive tests. One recent study reported a false positive rate of 52% in a small group (12 of 23) of control patients.[8] A pediatric study performed repeat tilt tests on 21 patients with typical vasovagal syncope on initial tilt testing. They reported that 67% of patients (14 of 21) experienced syncope or presyncope on repeat tilt after a 30 minute recovery period from the initial test. Neither specific symptoms reported, nor pattern of heart rate or blood pressure response was consistently reproducible.[9] The greatest benefit of tilt testing may lie in its ability to diagnose vasovagal syncope, precluding an extensive and costly work-up for other rare causes.

TREATMENT

If a diagnosis of vasovagal syncope is made, every effort should be made to educate and reassure the patient and parents of its benign nature. The conservative approach of reassurance, avoidance of known triggers, and lying down during prodromal symptoms will reduce or eliminate symptoms in the majority of patients. If conservative therapy is unsuccessful, pharmacologic therapy may be indicated. Beta-blockers may seem counter-intuitive, but they work by blunting the body's response to increased adrenergic tone caused by venous pooling. In those prone to syncope, increased adrenergic tone results in a physiologic overshoot response of complete sympathetic withdrawal and vagal excess which utlimately results in loss of consciousness. Important side effects such as depression and inattentiveness need to be monitored closely in the adolescent.

Patients who show a tendency to exaggerated orthostatic changes, or who have low baseline blood pressure may benefit from increased fluid intake aimed at increasing circulating arterial volume. This therapy may also be effective in patients with high vagal tone and low resting heart rate in whom beta-blockers are contraindicated. Fluid intake may be increased either by behavior modification, or the use of salt pills, salty snacks or prescribed mineralocorticoids to induce thirst. These modalities generally have excellent success. Fludrocortisone is generally used in a dose of 0.1-0.2 mg/day.

Patients with clearly identified cardiac anomalies will require aggressive intervention, Patients with HOCM will usually go on to some combination of surgery an / or implanted cardioverter to treat episodes of ventricular tachycardia. Those with coronary anomalies usually require surgical re-implantation of the aberrant artery.

To date, no randomized study has been performed in children to compare therapeutic efficacy between pharmacologic and conservative therapy. One controlled trial of tilt induced syncope has been performed in adults, comparing placebo to varied medical therapies, (primarily beta-blocker).[10] After 20 months of follow-up, resolution of symptoms was seen in 67% of the placebo group and 70% of the treatment group.

It is sometimes unclear which patient should be referred for evaluation by a pediatric subspecialist. Table 2 lists the historical and physical findings that should prompt a referral to either the pediatric cardiologist or neurologist. All patients who have syncope during exertion should be evaluated by a cardiologist.

SUMMARY

The optimal approach to the adolescent with syncope relies on a good working knowledge of its varied etiology; specifically, knowledge of the most common and generally benign type, vasovagal syncope, as well as the important distinguishing characteristics of the more serious and life threatening forms of cardiac syncope. A thorough history and physical examination are the cornerstones of investigation and will suggest the diagnosis in the vast majority of cases. Screening electrocardiography complements the history and physical, and is important in ruling out symptomatic arrhythmias impossible to diagnose by history and physical alone. Extensive invasive testing is usually unnecessary. Vasovagal syncope generally responds to reassurance and avoidance of precipitating triggers and positional changes. Beta blocker therapy is usually the first line of pharmacologic therapy. Treatment for other forms of syncope should be directed at the suspected underlying cause.

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THE CREATIVE CLINICIAN: CASE OF THE MONTH

"The practice of medicine is an art, not a trade; a calling, not a business ..." – WILLIAM OSLER, Aequanimitas

Editor: Anthony Mega, MD; Co-editor: Fred J. Schiffman, MD

Chronic Leg Pain in an Adolescent Male

William S. Ferguson, MD

An eleven year old boy was referred for evaluation because of a one-year history of intermittent right thigh pain. The pain originally started following a fall from a jungle gym. He was seen by his pediatrician, and had a normal physical examination and normal X-ray of the lower extremity. The pain initially improved, but then would return every 3-4 weeks. These episodes - which often followed minor trauma - would last for approximately a week, were intense enough to awaken him from sleep and to cause a limp, and were partially relieved by acetaminophen or ibuprofen. He had no fevers or night sweats, no other sites of discomfort, and no weight loss. He remained physically very active.

One week prior to presentation he had a routine visit to his pediatrician. At that time he was free of pain, but on physical exam his right thigh was very tender to palpation. There was no palpable mass, no limp, and he had normal range of motion at the hip and knee. An X-ray of the femur showed cortical thickening of the diaphysis with some periosteal new bone formation and an ill-defined lucency in the mid-shaft; no soft tissue mass was apparent (Figure 1). A bone scan showed increased activity in the femur, but otherwise was normal for age.

During the intervening week he remained asymptomatic. On physical exam, he was a well-developed, well nourished male (height = 75th percentile, weight = 95th percentile). HEENT, lung, heart, and abdominal exams were all normal. Examination of his extremities failed to reveal any palpable masses, and he was no longer tender even to deep palpation. His gait was normal, as was his neurologic exam.

An MRI of the lower extremity showed an abnormal medullary signal extending from the intertrochanteric region to 11cm above the distal end of the femur. There was expansion of the medullary space with endosteal scalloping and periostitis, but no definite extension through the cortex and no soft tissue mass (Figure 2a and 2b). This was felt to be most consistent with chronic osteomyelitis; eosinophil granuloma and sarcoma were also considered possible.

Abbreviations Used:

CT	computed tomography
FISH	fluorescent in-situ hybridization
H&E	hematoxylin and eosin
HEENT	head, ear, eyes, nose, throat
MRI	magnetic rssonance imaging
PCR	polymerase chain reaction
PNET	primitive neuroectodermal tumor

An open incisional biopsy of the femur was performed. Cultures of the material were negative; histologic examination showed a small, round blue-cell tumor. Immunohistochemical testing showed that the tumor was positive for keratin, vimentin, and mic-2, and was negative for leukocyte common antigen, desmin, and neuron-specific enolase (respectively, markers for lymphoma, rhabdomyosarcoma, and neuroblastomas or other neuroepithelial-derived tumors). Electron microscopy showed aggregates of glycogen; evidence for neuronal differentiation was lacking. These results were diagnostic of Ewing's sarcoma.

A chest CT showed no obvious nodules, and a bone marrow aspiration/biopsy was negative for metastatic tumor. The patient started chemotherapy with vincristine, doxorubicin, and cyclophosphamide alternating with etoposide and ifosfamide at three-week intervals. Repeat radiographs after 4 cycles were essentially unchanged.

He then underwent surgical resection of the involved bone with placement of a bone allograft (Figure 3). Histologic examination of the tumor showed >95% tumor necrosis with only rare nests of viable-appearing cells, and all

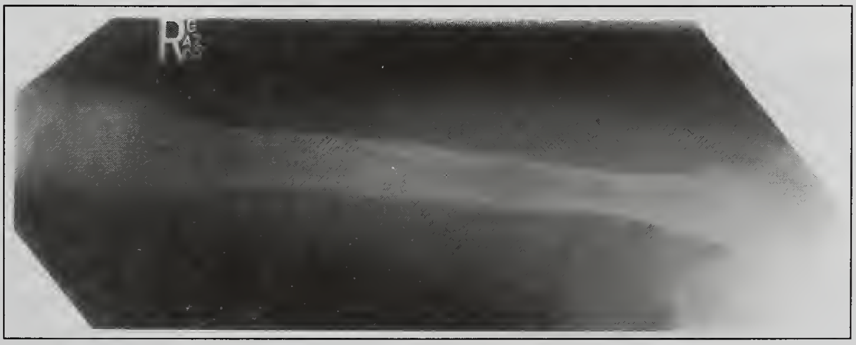
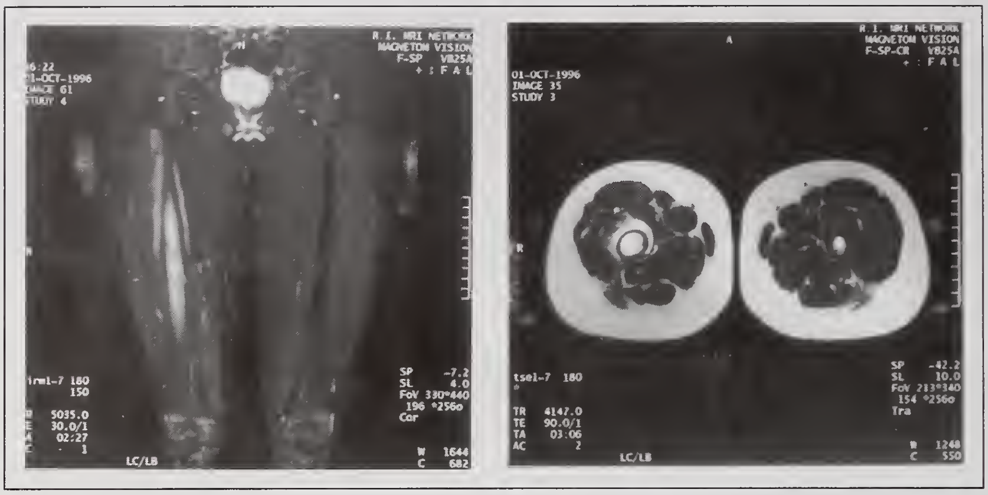


Figure 1.



Figures 2a and 2b.

resection margins were negative for tumor. He received an additional 13 cycles of chemotherapy after surgery. Other than the expected periods of pancytopenia, he tolerated chemotherapy well. His leg also showed progressive healing throughout the period of his chemotherapy.

As of his last visit, 30 months following his initial diagnosis, he was asymptomatic and without radiographic evidence for tumor recurrence. His bone graft showed extensive healing at both ends. His gait and activity level were normal.

DISCUSSION

Primary bone tumors are relatively rare, comprising only about 5% of childhood malignancies. About 60% of primary bone tumors will be osteosarcoma, and about one-quarter will be Ewing's sarcoma. Both tumors are more common in males, and both have a peak incidence during adolescence (although this age distribution is more striking for osteosarcoma, and during the first decade of life the two tumors are about equally common). Ewing's sarcoma is relatively rare among African-Americans and Asians. There is no known predisposing factor to the development of Ewing's sarcoma. About half of all cases will arise in the pelvis or axial skeleton, and when it does originate in the long bones it is most commonly in the diaphysis.

Pain is the most common presenting symptom. Fever, weight loss or other constitutional symptoms are less common but when present may erroneously suggest the presence of infection. A palpable mass and limitation of joint movement are the most common physical findings, although soft tissue masses may be difficult to detect in the thigh (particularly in a muscular teenager), and even large masses may be impossible to feel when arising in the pelvis or trunk. A long duration of symptoms does not exclude a neoplastic process, since often more than 6 months will elapse from first symptoms until a correct diagnosis is made. For these reasons, radiographic evaluation should be undertaken in any patient with chronic or recurrent unexplained bone pain.

Classically, bone tumors will show both bone destruction and sclerosis; a soft tissue mass is often seen. CT and MRI are more sensitive than plain X-rays in showing the degree of marrow involvement and the size of any soft tissue mass. Although the classic X-ray finding in Ewing's sarcoma is an "onion-skin" appearance resulting from periosteal reaction, calcification may be present which can be difficult to distinguish from the neoplastic new bone often seen in osteosarcoma. Thus in many cases it is impossible on clinical or radiographic grounds to distinguish between various tumors, chronic infection, and eosinophilic granuloma. In particular, the possibility of malignancy should be kept in mind when evaluating patients with presumptive osteomyelitis.

Material for histologic examination is required to establish a firm diagnosis; open biopsy is the usual procedure, although at times multiple needle biopsies will suffice. Small round blue-cell tumors that arise primarily from bone include Ewing's sarcoma, primitive neuroectodermal tumors, and lymphomas; metastatic lesions with this appearance include neuroblastomas and some rhabdomyosarcomas. It can be difficult or impossible to distinguish among these on the basis of conventional H&E stains. Immunohistochemical stains will often yield a firm diagnosis; in particular, expression of mic-2 strongly supports the diagnosis of Ewing's sarcoma or PNET in this context.

... radiographic evaluation should be undertaken in any patient with chronic or recurrent unexplained bone pain.

Ewing's sarcoma and primitive neuroectodermal tumors (PNETs) both frequently exhibit t(11,22) translocations which result in an aberrant EWS-FLI1 fusion protein. This protein is capable of inducing transformation in cell lines, and presumably plays some role in tumorigenesis. Although careful chromosome analysis will usually show the translocation, PCR or FISH analysis is technically easier and more sensitive in cases where immunohistochemistry is not diagnostic. In a minority of cases, a t(21,22) translocation is present instead. PNETs are distinguished from Ewing's sarcoma by the degree of neuronal differentiation; however, they have very similar biologic behaviors and response to treatment, and so now are generally considered variants of the same tumor.

Although only 20-25% of patients will have obvious metastatic disease at diagnosis (most commonly to lung, but also to bone and bone marrow), treatment with surgery alone almost always results in distant relapse. This implies the almost universal presence of micrometastatic disease and is the rationale for adjuvant chemotherapy. The most commonly used drugs include vincristine, actinomycin-D, doxo-

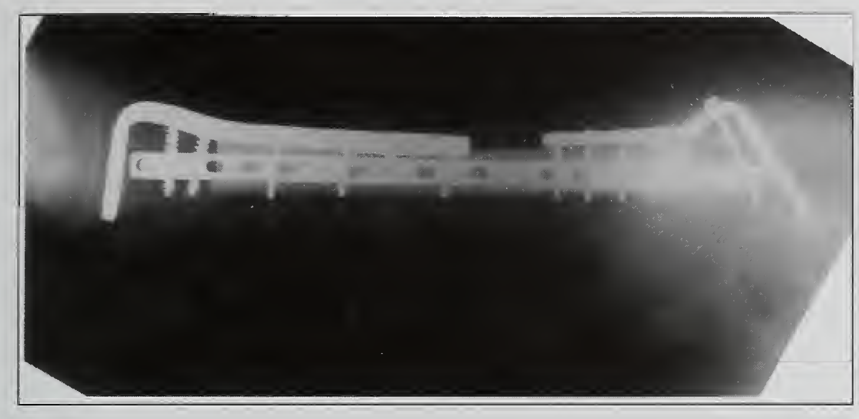
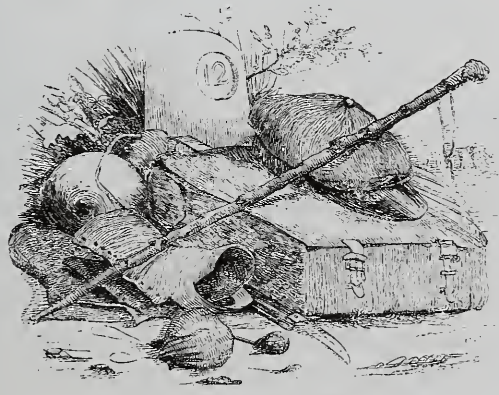


Figure 3.

rubicin, and cyclophosphamide. Improved survival (particularly in high-risk patients) has resulted from the addition of etoposide and ifosfamide and utilization of more intensive dosing regimens.

Ewing's sarcoma typically shows a good initial response to chemotherapy, often with near-complete resolution of the soft-tissue mass and extensive necrosis of tumor within bone. Although chemotherapy is not considered sufficient treatment for large tumor masses, a good initial response to chemotherapy may allow greater choice of local control measures. Unlike osteosarcoma, Ewingís sarcoma is considered to be radiation-sensitive, so treatment of the primary tumor with radiation is a feasible option in cases where surgical removal would cause functional or cosmetic problems. However, radiation is not without potential long-term adverse effects (including damage to normal tissues and secondary sarcomas). Thus, surgery is being used more often, particularly as improved techniques have allowed good functional outcomes following limb-salvage procedures.

Although size and location of tumor have prognostic significance (large and proximal tumors doing less well than small or distal tumors), some of these differences have disappeared with improved therapy. Children have a better outcome than older adults, and the overall survival of patients who present without overt metastases is now 60-70%. Treatment of patients with metastatic disease remains disappointing; although patients with metastases confined to the lung do better than those with disease involving the bone or bone marrow, the overall survival is still only 20-25%. A small proportion of patients with recurrent tumor can be salvaged, usually with some combination of surgery, radiation, and high-dose chemotherapy.



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Advances in Pharmacology

Update on the Use of Oral Contraceptive Pills for Emergency Contraception

Patricia J. Flanagan, MD

Adolescent pregnancy rates in the US have declined more than 17% during the last decade. This is a significant success. Still, the US leads all other industrialized nations in rates of pregnancy and childbearing among its adolescents. We still have 97/1000 girls 15-19 years old becoming pregnant each year. The majority of these pregnancies are unplanned, and greater than 40% end in terminations. As health care providers who care for teenagers, we can do more to prevent unwanted pregnancies. Emergency contraception offers women who have had unprotected intercourse a second chance at avoiding unwanted pregnancy.

Emergency contraception is not a new pharmacologic intervention. It has been available in various forms since the late 1960s. Some recent developments, however, make the use of this safe and effective means of hormonal postcoital contraception more widely accessible. Adolescent health care providers need to understand this form of contraception, how and when to prescribe it, and especially the importance of educating our patients and the public at large on its availability. People will not seek it out and ask for it if they are unaware of its existence. It is safe, it is FDA-approved, and although it is a postcoital method (taken after intercourse), it is a contraceptive method, not an abortifacient. It prevents pregnancies from occurring and will not terminate existing pregnancies.[1,2]

> *For women taking the Yuzpe method within 72 hours of unprotected intercourse, the risk of pregnancy is reduced by 70-80%.*

In 1969, a 5-day course of high-dose estrogen commonly known as 5 x 5 was shown to be a highly effective means of preventing pregnancy when given within 72 hours after unprotected intercourse.[3] Its side effects, although not dangerous, were common with 40-70% of women experiencing nausea and vomiting.[1] This method prevented pregnancy by preventing ovulation when given early in the menstrual cycle and by preventing the implantation of a fertilized ovum when given late in the cycle. Because of its side effects, this method fell out of use when other alternatives became available.

In 1974 Dr. Albert Yuzpe published studies on the efficacy of combination estrogen and progestin products in postcoital contraception.[4,5] Use of combined estrogen with progestins required much lower doses of estrogen. The treatment consists of 200 mcg ethinyl estradiol and 2.0 mg norgestrel divided into 2 doses, 12 hours apart. It is given within 72 hours of unprotected intercourse. This regimen, known as the Yuzpe Method, is still the most widely used method in the United States. The most common side effects are nausea and vomiting although at much lower levels with the 5 x 5 regimen.[1] This method most likely prevents pregnancy in the same manner as high dose estrogens; that is, early cycle ovulation is prevented and late cycle implantation is prevented. It also thickens cervical mucus, shortens the luteal phase, alter tubal transport time, and suppresses endometrial hormone receptors and function.[1,6,7] It has been widely used internationally for many years. However, its use in the US has been, until recently, limited to emergency room treatment of rape victims. Although not recommended to be given during pregnancy, its inadvertent use during pregnancy has not had reported teratogenic effects.[8] Its contraindications are the same as those of combination OCP's in general. Its use is contraindicated in patients with clotting disorders, heart disease, and history of thromboembolic disorders.

The issue of efficacy is complex due to varying risk with cycle time. The likelihood of a single act of intercourse resulting in pregnancy depends on its temporal relation to ovulation and ranges from almost zero to 33%. For women taking the Yuzpe method within 72 hours of unprotected intercourse, the risk of pregnancy is reduced by 70-80%. Pregnancy rates among women who used the method are 1.8- 2.8%.[1,9]

High-dose progestins have been the most recent addition to postcoital pharmacological emergency contraception. Levonorgestrel 0.75 mg, 2 doses 12 hours apart recently has been shown to be a more effective than the Yuzpe method and with fewer side effects.[9] Until recently the only progestin-only pills currently available in the US were very low dose pills containing 0.075 mg norgestrel (equivalent to 0.0375 mg levonorgestrel) per pill with a total of 40 pills needed (20 pills initially and 20 pills 12 hours later). Recent approval of newer formulations may make this a promising alternative to Yuzpe and it is anticipated that there will be a 0.75 mg preparation of progestin available on the US market soon.

The timing of postcoital contraception has been studied widely with varying results. It seems that combination OCPs and progestin only pills are most effective when taken within 24 hours of unprotected intercourse. They are, however, very effective when taken as far as 72 hours after intercourse.[9-12] The efficacy rates of 70-80% are from women taking pills up to 72 hours post-intercourse.[9]

In 1997 the FDA officially approved the use of OCPs for postcoital contraception.[13] The agency named 11 different products and dosages for the addition of this indication. Three of the most commonly used products were Ovral (2 pills every 12 hours x 2 doses), LoOvral (4 pills every 12 hours x2 doses) and Nordette (4 pills q 12 hours x 2 doses). In an aggressive public awareness campaign a non-profit organization, "Bridging the Gap," offers a toll-free phone number to help patients locate a provider in their area who will provide prescription for postcoital contraception. (888-not-2-late). The Office of Population Research at Princeton University maintains a website with patient information and office posters (http:opr.princeton.edu/ec/ec.html).[14]

In September 1998 the first emergency contraceptive kits became available in the US. These are marketed by Gynetics under the name of Preven. The 4 pills in the Preven emergency Contraception Kit are combination oral contraceptives. Each pill contains 0.25 mg levonorgestrel and 0.05 mg ethinyl estradiol. The kit also contains a urine pregnancy test (a monoclonal antibody test for hcg). The patient is instructed to check the urine pregnancy test for pre-existing pregnancy prior to taking the 2 pills followed 12 hours later by the remaining 2 pills. The kit is for use within 72 hours of unprotected intercourse.

In August 1999 a preparation of 0.75 mg levonorgestrel received FDA approval. Soon to be available under the name "Plan B," each kit will contain 2 pills, the first to be taken with 72 hours of unprotected intercourse, the second to be taken 12 hours later.

Take advantage of missed opportunities. In order for emergency contraception to be used effectively, women need to know about it and need to have access to it in a timely manner.[14,15,16] As health care providers we must make sure patients are aware of this option to unwanted pregnancy so that they can ask for it. Some health care providers supply information, including written advice, regarding postcoital contraception at health maintenance visits to all adolescents of childbearing age. Others supply a prescription for Preven Emergency Contraception Kit or OCs to sexually active patients and to those contemplating sexual activity. Many providers take the opportunity of postcoital contraception visits to educate patients regarding healthy sexual decision making.[14] While safe and effective in preventing unwanted pregnancies, emergency contraception does not protect against STD or HIV infection. Discussions regarding risk of STDs and use of condoms are warranted. For adolescents who are sexually active on an ongoing basis, it provides the opportunity to discuss hormonal contraception such as depo-provera, norplant, or OCPs, or IUDs or barrier methods. When treating young women with emergency contraception, some providers schedule a return visit 2 weeks following the administration of the pills. This visit offers the opportunity to test for pregnancy (treatment failure), to screen for STDs, and to re-enforce education.

Emergency contraception is a safe and effective hormonal way of preventing unwanted pregnancy. Its role in this country, unfortunately, has been limited to college health clinics and emergency rooms following rape. There is no reason this method should not be used more widely. We, as health care providers to adolescents, can educate our patients and the public at large to its availability in an effort to further reduce the rates of unwanted pregnancy among our adolescent patients.

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Point of View: Why We Need School Based Health Centers in Rhode Island?

Rosemary Reilly-Chammat, MPA

SBHCs are primary health care settings located in schools. They provide acute care, such as treatment of colds and injuries; chronic care for conditions such as asthma and diabetes; mental/behavioral health services, including substance abuse services; yearly physical and sports examinations; reproductive health care including diagnosis and treatment of sexually transmitted diseases; dental care; nutrition services; and immunizations. The first SBHCs were established in the early 1970s.[1] During this past decade SBHCs grew to 1,157 nationwide. Most SBHCs (63%) are in urban schools; 26% are in rural schools; 11% in suburban schools. Thirty-seven percent (37%) are in high schools, 34% elementary schools, 16% in middle schools, 7% in other schools, and 6% in K-12 schools. (Figure 1)

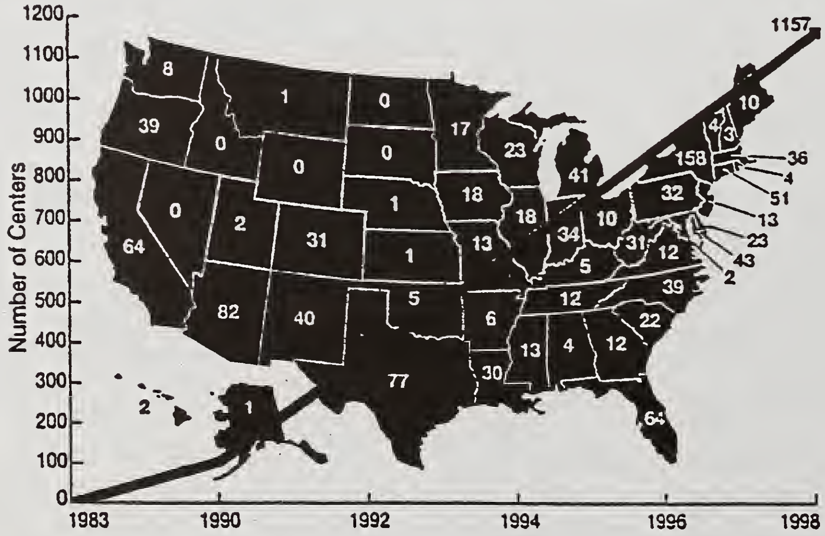


Figure 1. Data are from the 1998 National Survey of State School Based Health Center Initiatives. Reprinted with permission from *Making the Grade*, George Washington University Medical Center, Washington, D.C.

Abbreviations Used:

SBHC	School-Based Health Center
PCP	Primary Care Provider
COZ	Child Opportunity Zone

RI Perspective

Under RIte Care, the State of Rhode Island has made health insurance available to children. However that alone does not guarantee entry into the health care system. During early childhood, families see that children are up-to-date on the immunizations and preventive care services they need to enter school. Afterward, attention to preventive health care can wane, particularly for adolescents. SBHCs bring physical health and behavioral health services to students who need them. Staff encourage teens to include family members in health care decisions as appropriate.

Rhode Island's school-based health centers are based in schools with a high proportion of low-income teens:

- Providence's Central High School, operated by Providence Health Center, Inc. since 1987
- Woonsocket High School and Middle School, operated by Thundermist Health Associates since 1998 and 1993 respectively
- Central Falls Jr./Sr. High School, operated by Blackstone Valley Community Health Care since 1995
- Pawtucket's Slater Middle School, operated by Memorial Hospital, since 1999. Pawtucket's Cunningham Elementary School is operated by this SBHC also.
- Pawtucket's Shea High School and Woonsocket's Coleman Elementary School opened this fall.

Nurse practitioners provide the majority of services, with medical consultation from a physician once a week on site and as necessary. Behavioral health providers address mental health and substance abuse needs. All SBHCs are operated by a licensed health facility that provides after-hour coverage at community sites. (Figure 2)

Students who use the SBHC must have a signed parental consent on file. For some students, the SBHC is their only regular source of care. The sponsoring health provider is the student's PCP. If SBHC users have a PCP in the community, the SBHC coordinates care with that PCP. The SBHCs are attractive for parents or guardians who have jobs that are difficult to leave, insufficient transportation and /or inadequate child care for younger siblings. (Figure 3)

SBHCs operate where student needs are greatest, provide services re-

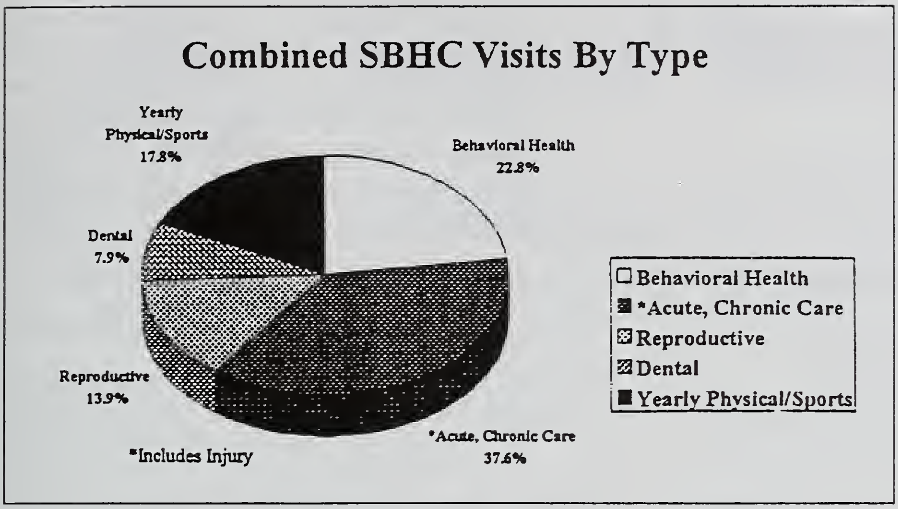


Figure 2. Rhode Island SBHC data are from the 1997-98 school year.

gardless of ability to pay, offer confidential care per state and federal statues and have expertise in adolescent health. RI needs SBHCs because adolescents in our urban communities are at increased risk for every Kid's Count health and education indicator of need.[2] Further, national studies have shown that SBHCs can improve dropout rates and absenteeism.

Consider the following:

- The cumulative drop-out rate in selected urban communities ranges from a high of 49% to a low of 8%.
- The 35,000 suspensions for Rhode Island involved only 1,300 students. One third of all suspensions occurred in 11 urban communities.
- More than three quarters of uninsured children in Rhode Island live in families that are eligible for RIte Care; 4,800 children live in families that do not meet the income requirement for RIte Care and are uninsured. As of December 1998, 88% of all children, under 18, enrolled in RIte Care, reside in 11 urban communities.
- Many children are affected by mental health problems. National studies show that at least 1 in 5 children and adolescents may have a mental health problem, and 1 in 20 may have a serious emotional disturbance.
- Rhode Island's urban communities have the highest rates of teenage pregnancy, STDs, tobacco use, alcohol and other substance abuse, and intentional and unintentional injuries, compared to Rhode Island's suburban and rural communities.

VISION AND OPPORTUNITY

Over the next two years, Rhode Island, with funding from the Robert Wood Johnson Foundation's Making the Grade Program, has an opportunity to develop and sustain SBHCs. The Department of Health's vision is to establish SBHCs in urban high schools initially, followed by urban middle schools. The Department is engaged in discussions with state and community stakeholders on a strategy.

FITTING SQUARE PEGS IN ROUND HOLES

SBHCs are often viewed as square pegs in round holes. RIte Care health plans are required to include SBHCs in their networks and to reimburse SBHCs for the services they provide. SBHCs have been able to recoup about 20% of billable expenses, leaving unreimbursed a sizable portion of operating expenses. The majority of teens who use SBHCs visit three or more times per school year. Further, some private insurers do not cover services offered through schools and or colleges.

SBHCs are designed to meet the needs of students within a particular school. While there is a common core of services, the configuration of services may vary by school. SBHC staff need to work with school and community providers to overcome turf issues and in concert, form an integrated safety net of services.

Reimbursement for behavioral health services has been problematic due to local provider practices clashing with managed care standards for credentials. Providers, health plans and state agencies need to work together to assure that all students have access to quality behavioral health services at SBHCs.

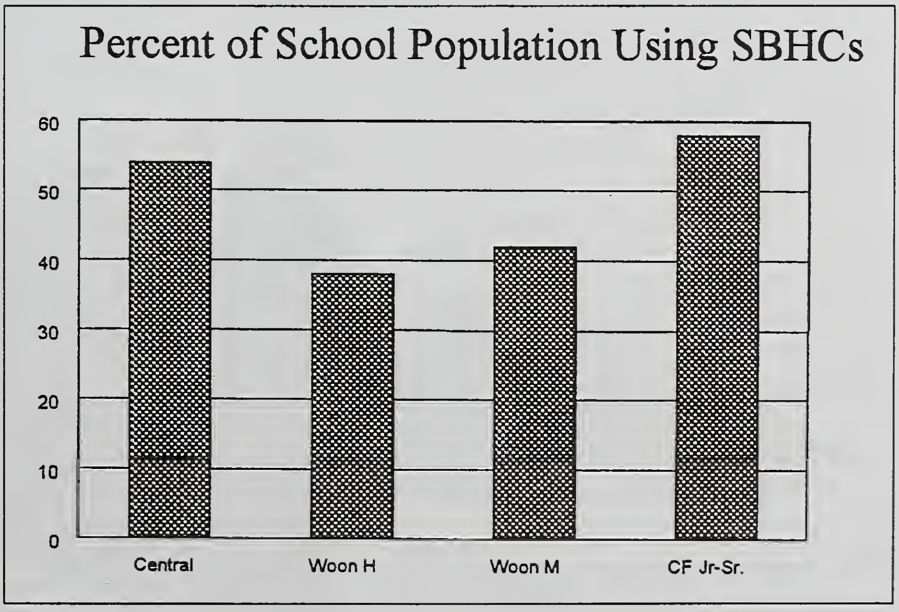


Figure 3. Rhode Island SBHC data are from the 1997-98 school year.

COMPONENTS OF A COMPREHENSIVE SCHOOL HEALTH PROGRAM

✓ NUTRITION
✓ PHYSICAL EDUCATION AND ACTIVITY
✓ HEALTH EDUCATION
✓ HEALTH SERVICES
✓ COUNSELING, PSYCHOLOGICAL AND SOCIAL SERVICES
✓ HEALTHY SCHOOL ENVIRONMENT
✓ HEALTH PROMOTION FOR STAFF
✓ FAMILY AND COMMUNITY INVOLVEMENT

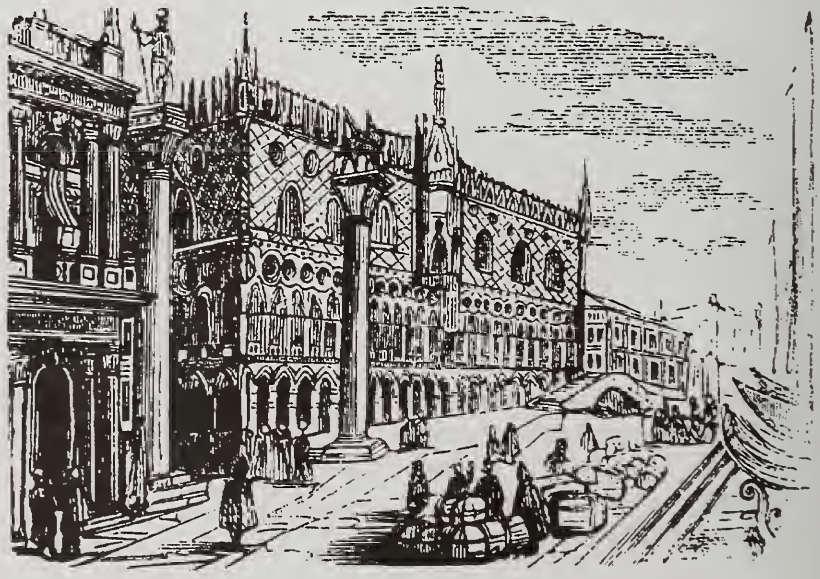
CONCLUSION

SBHCs add value to schools because the SBHCs help students get down to the business of learning. SBHCs bring public health benefits to schools by providing ongoing social and behavioral support for positive health behaviors. SBHCs help adolescents continue ongoing productive relationships with health care providers as the adolescents leave school and enter young adulthood.

We hope we can count on your support for school-based health services. As physicians in the community, you can show your support by:

- Visiting SBHCs in your community to learn about the services they provide and how you can work together to address adolescent health concerns.
- Contacting schools in your community to see if they are considering SBHCs and offer support.
- Participating in the planning and ongoing advisory to SBHCs in your community.
- Sharing your expertise in child/adolescent health to enable all school and community members to value the critical relationship between health and educational success.

Rhode Island's children's policy aims include: all children enter school ready to learn, all youth leave school prepared for productive lives, and all children are safe in their homes and communities. The Rhode Island Children's Cabinet (the Departments of Health; Education; Human Services; Children, Youth, and Families; and Mental Health, Retardation, and Hospitals) is developing strategies to help local school departments support comprehensive services. Child Opportunity Zones (COZs) offer preventive services mainly for early school years. Youth services provide after-school care, including preventive services. When SBHCs, COZs, youth services and other family support systems are linked, they form comprehensive systems of care for teens, children and their families.



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Judicial Diagnosis

Law School for Physcians: Consent by Minors

Jeffrey Wishik, MD, JD

Abbreviations Used:	
DOJ	Department of Justice
FTC	Federal Trade Commission

"Every human being of adult years and sound mind has a right to determine what shall be done with his own body." Justice Benjamin Cardozo[1]

This widely-cited statement by the eminent jurist Cardozo is generally regarded as the cornerstone of the traditional approach to consent in medical practice. Rhode Island law establishes eighteen years as the age of consent.[2] At that age, an individual can give consent and is entitled to confidential treatment - even if he or she is living with parents who are paying the bills. Needless to say, the physician may be placed in an uncomfortable position between a parent accustomed to making decisions for a child and the new "adult" who insists that the parent not be involved. This might be avoided by starting to involve the minor in decision-making at a younger age while encouraging parents to allow greater autonomy and privacy. In fact, this is the recommended approach of the American Academy of Pediatrics - obtaining patient assent and parental *informed permission* for younger children.[3]

The general rule is that parents have the power to consent for minor children. (Other caregivers cannot give consent unless they have parental authorization, preferably in writing.) However, physicians must be wary of parents attempting to force treatment - such as psychiatric hospitalization - on their children for punitive purposes. Also, additional state and federal laws extend the right to consent to certain minors based either on their legal status or the existence of particular medical conditions. Thus, parents may not hold absolute power when it comes to providing consent.

For example, most clinicians probably know of the exception for *medical emergencies.* Legally, courts find implied consent in these situations, similar to the rationale allowing treatment of comatose adults. That is, the court presumes that if the parent or guardian had known of the imminent risk to the child's health, s/he would have authorized treatment. The emergency must be an existing condition, not merely a potential one, but it need not be life-threatening. Certainly, treatment should not be delayed if waiting for consent poses a clear risk to the child's well being.

"*Mature minors*" may also have the right to provide their own consent. Both the definition of maturity and the extent of this right vary from state to state. In Rhode Island, married persons and anyone sixteen years or older may consent to "routine emergency medical or surgical care."[4] The statute does not define the seemingly incongruous phrase "routine emergency." Presumably, treatment for broken bones, lacerations, infections and the like is covered. On the other hand, organ transplantation probably would not be deemed "routine." Interestingly, this law can lead to the unusual situation where fifteen year-old single parents would not be able to provide their own consent for emergencies, but could provide it for their children.

Emancipation is another exception to the rule of parental consent based upon the minor's legal status. In some jurisdictions, emancipation occurs automatically through

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marriage or active military service; in others, a statute specifies requirements. Rhode Island does not have a law defining emancipation. Instead, a judge must rule on a minor's petition for emancipation, making the decision based upon the individual's circumstances, needs and maturity. Physicians should remember that the *mature minor* and *emancipation* exceptions depend on whether legal standards of capacity for consent have been met. It is not the physician's judgment that is determinative.

Public health and social policy concerns have led to laws in every state that allow minors to receive treatment without parental consent for specified conditions. Family planning, substance abuse and infectious diseases are the usual areas covered. Thus, in Rhode Island minors may consent to "testing, examination and/or treatment for any reportable communicable disease."[5] The covered conditions include HIV, sexually transmitted diseases, tuberculosis and a wide range of other infections or infestations.[6]

> *Contraceptives may be distributed to minors under Title X without parental consent or notification.*

Many states permit minors to receive substance abuse treatment without parental consent or notification. If a minor refuses to allow parental contact, Rhode Island law allows noninvasive and noncustodial treatment for substance abuse and chemical dependency. However, during treatment the statute urges the clinician to attempt to obtain the minor's permission to contact the parents.[7] However, federal statutes and regulations may supercede the state law. If a treatment program receives any direct or indirect federal assistance its clients - *including minors* - are guaranteed strict confidentiality.[8] Evaluation, treatment and counseling are covered; confidentiality applies from the moment the client applies for or even inquires about the program. The federal law and its implementing regulations do not mention "consent." However, by requiring strict confidentiality the law implicitly grants the power of consent to the minor.

The federal family planning services grant program, Title X, also covers "unemancipated minors who wish to receive services on a confidential basis,"[9] without regard to age or marital status.[10] Contraceptives may be distributed to minors under Title X without parental consent or notification. Efforts to require parental notification by a federally funded program have been denied by the courts.[11] Note, however, that abortion services are not covered by Title X. Rhode Island requires parental consent for a minor's abortion unless an appropriate court either finds her to be emancipated or determines that other statutory exceptions to notification are present.[12]

Divorce raises other problems for the treating physician. Generally, only one parent's consent is needed for treatment. It is possible for a divorce decree to specify which parent has the power to consent to a minor's treatment. However, at least in Rhode Island, this is rarely done. In the typical joint custody arrangement both parents have this power, regardless of which parent has physical custody of the child. Problems can arise if the parents disagree about a medical course of action. Particularly when dealing with elective treatment the physician should avoid seeking consent from one parent after the other has denied it. If the dispute cannot be resolved easily, the physician may need to delay non-emergent care until the parents find a solution or bring the matter to court.

If one parent has sole legal custody the other cannot provide consent. Unfortunately, without a copy of the divorce decree or custody agreement the physician can incorrectly assume that the parent purportedly giving consent has that right. Therefore, physicians caring for minors should inquire about custody and consent. If need be, copies of divorce decrees can be placed in the child's medical chart. Similarly, a custodial parent should be asked to provide written proxy consent either for the noncustodial parent or other caretakers. This facilitates necessary but non-emergent treatment of the child when the custodial parent is unavailable. By anticipating consent problems the clinician should be able to avoid or minimize these difficulties.

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Health by Numbers

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Jay S. Buechner, PhD

Divergences in Smoking Patterns Among Younger and Older Rhode Islanders, 1988 and 1998

Jana E. Hesser, PhD

The 23th annual Great American Smokeout will occur on November 18, 1999. While the prevalence of smoking among U.S. adults declined steadily from the mid 1960s through the 1980s, the decline slowed in the 1990s. An estimated 47 million adults in the U.S. (about 23% of adults ages 18 and over) smoke cigarettes, even though smoking cigarettes will result in death or disability for half of all regular users.[1] Approximately 80% of adult smokers started smoking before age 18.[2] Decreasing the number of cigarette smokers in the U.S. and preventing the initiation of smoking among young people remain major public health challenges.

This paper compares patterns of cigarette use among Rhode Island adults in 1998 with use patterns in 1988 based on data from Rhode Island's annual Behavioral Risk Factor Surveillance System (BRFSS).

Methods

The BRFSS is a national telephone survey of randomly selected adults (ages 18 and older) who live in households with telephones. It asks respondents questions about a variety of health related behaviors, including tobacco use. The BRFSS is performed by 50 states and 4 territories with funding and methodological standards provided by the Centers for Disease Control and Prevention (CDC).[3]

The BRFSS has been conducted in Rhode Island continuously since 1984, and by a professional survey contractor since 1990. In 1988, 1763 Rhode Island adults were interviewed, or approximately 150 per month. In 1998 the sample size was increased to 3,602, with 300 interviews conducted each month.

Key questions on tobacco use in the BRFSS have remained unchanged or been modified only slightly between 1988 and 1998, so that respondents can be categorized across years as follows:

- Current Smokers: Have smoked 100 or more cigarettes during their life, and smoke now.

Abbreviations Used:

CDC	Centers for Disease Control and Prevention
BRFSS	Behavioral Risk Factor Surveillance System

Current smokers can further be categorized as:

- Current Regular Smokers, that is, persons who now smoke every day, and
- Current Irregular Smokers, that is, persons who now smoke but not every day.

- Former Smokers: Have smoked 100 or more cigarettes during their life but no longer smoke.
- Never Smoked: Have not smoked 100 or more cigarettes during their life.

In addition, current regular smokers can be categorized by the number of cigarettes usually smoked each day.

Results

The prevalence of current smoking (which includes both regular and irregular smokers) decreased from 26.0% to 22.6% between 1988 and 1998. (Figure 1) The proportion of current smokers who are regular (everyday) smok-

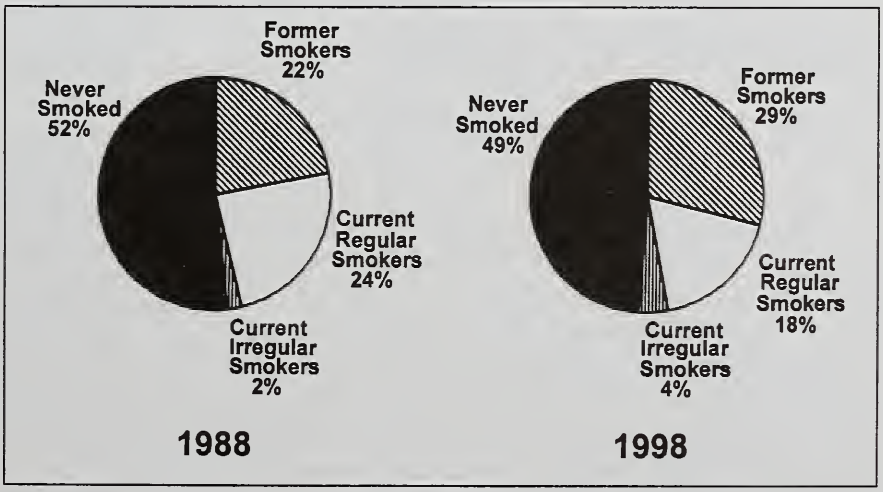

Figure 1. Smoking Patterns among Rhode Island Adults Ages 18 and Older, 1988 and 1998.

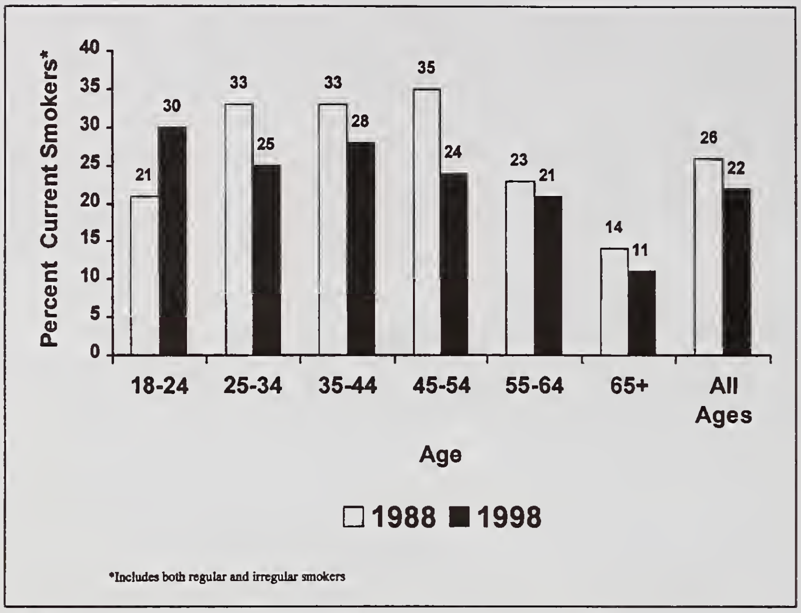

Figure 2. Current Smokers* among Rhode Island Adults by Age, 1988 and 1998.

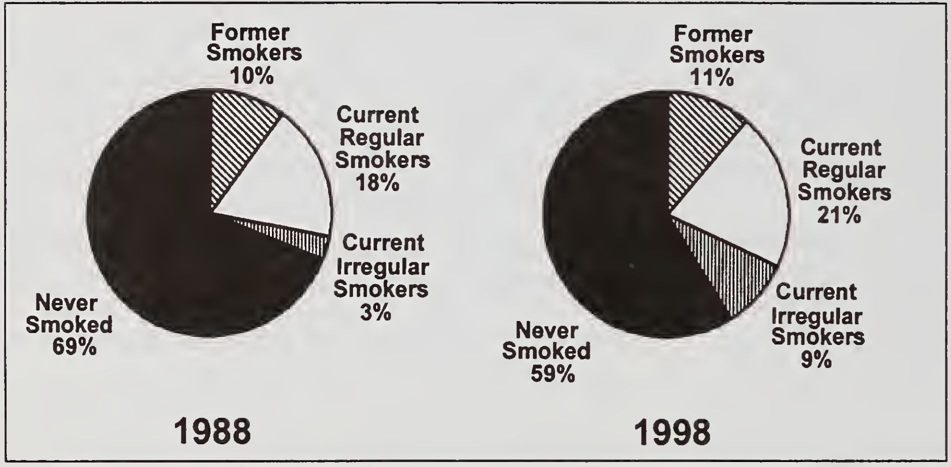

Figure 3 Smoking Patterns among Rhode Island Adults Ages 18 - 24, 1988 and 1998.

ers dropped from 92% to 82%, while the proportion who are irregular smokers increased.

The prevalence of current smoking decreased among persons ages 25 and older. However, among those ages 18 - 24 the prevalence of current smoking increased from 21% to 30%. (Figure 2) The prevalence of both current regular and irregular smokers in this age group has increased, but the greater increase (from 14% to 30% of current smokers) occurred among irregular smokers. (Figure 3) The increase in smoking among young adults parallels trends of increased smoking prevalence among adolescents observed in national and state studies over the past decade.[4,5]

The decline in adult smoking rates can be attributed entirely to adults who have quit smoking, rather than to a decline in smoking initiation. The proportion of adults who are former smokers increased from 22% to 29%, while the proportion who never smoked decreased, from 52% to 49%. (Figure 1) Among those ages 18-24 the proportion who are former smokers has remained virtually unchanged (10% and 11%) while the proportion who never smoked decreased from 69% to 59%. (Figure 3)

Among current regular smokers ages 25 and older, it appears that an increasing proportion are heavier smokers who may find it very difficult to quit smoking. The proportion who smoke more than 1 pack of cigarettes a day increased from 24% in 1988 to 27% in 1998. Among those 18 -24, the proportion smoking more than 1 pack per day is much lower (10% in 1988 and 8% in 1998).

Discussion

These data indicate clearly that the tobacco industry has been successful over the past decade in recruiting new smokers among young people. The increased smoking prevalence among young adults has partially offset the successes of smoking cessation campaigns and programs among adults ages 25 and older. Further reductions in adult smoking prevalence will require increased efforts to prevent smoking initiation among adolescents and young adults, as well as smoking cessation efforts to help heavier smokers quit.

Jana E.. Hesser, PhD, is Health Policy Analyst, Office of Health Statistics, and Coordinator for the Rhode Island Behavioral Risk Factor Surveillance System.

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PUBLIC HEALTH BRIEFING

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

DEARS, DOES and Diabetes

Joann M. Lindenmayer, DVM, MPH, and Dona L. Goldman, RN, MPH

Abbreviations Used:

DCCT	Diabetes Control and Complications Trial
DOE	Diabetes Outpatient Educator
DEARS	Diabetes Education, Assessment, Referral, and Screening

Physicians' expectations for diabetes have recently, and dramatically, shifted. Physicians used to expect the course of diabetes to include a series of acute clinical complications. The 1993 Diabetes Control and Complications Trial (DCCT) changed that. The DCCT demonstrated that close, careful management could delay or prevent those acute complications. Based on these results and the development of national standards of diabetes care, health care systems are using disease management models and outcome measures. Diabetes management, particularly in light of the DCCT results, is time-consuming and labor-intense for physicians and patients alike. Physicians, constrained by their "managed care" plans' regulations, may find it difficult to adhere to these newer diabetes standards of care.

To address these barriers, the Diabetes Control Program of the Rhode Island Department of Health has entered into a partnership with Harvard Pilgrim Health Care of Rhode Island and Rhode Island Quality Partners, to create, develop, and implement the Diabetes Education, Assessment, Referral, and Screening ("DEARS") Program. Funded by the CDC, DEARS is based on a model developed by Kaiser Permanente and more recently applied in Rhode Island by Harvard Pilgrim Health Care. DEARS brings together a professional team with a group of patients who have diabetes. The team includes a physician, nurse, dietician, pharmacist, and - ex officio - the patient's physician. This team approach enables members to complement and reinforce diabetes management messages delivered by the physician and enables patients to receive comprehensive diabetes management in a single visit. The program is targeted at patients with diabetes who are 65 years of age or older (more than half of all persons with diabetes in Rhode Island). The DEARS program is particularly appropriate for this group for several reasons. Research indicates that older adults are comfortable with traditional classroom settings, desire a high level of social interaction, have a marked preference for in-class discussions, and prefer course content to be relevant to their own experiences.[1] All of these features of a successful learning environment for older adults are present in the DEARS program.

The program is organized as follows. A physician, within his/her office practice, identifies patients with diabetes and invites them to attend a DEARS session. Several days before the session, the DEARS staff abstract the medical records of patients who accept this invitation onto a form that:

- Provides the physician with a short summary of the patient's diabetes-related health problems and dates when the patient last received the following:
    - foot examination
    - lipid profile
    - HbA1c test
    - vaccinations for influenza and S. pneumoniae
    - dilated eye examination
    - diabetes self-management education.
- Provides the DEARS pharmacist with a synopsis of the patient's prescribed medications, and
- Provides a format in which to record diabetes-related clinical preventive services, tests, and referrals that the patient receives on the day of the DEARS program.

The physician reviews this synopsis and decides which laboratory tests, vaccinations, and referrals for additional examinations each patient should receive on the day of the DEARS program.

A typical DEARS session will include 10 patients and take place at that

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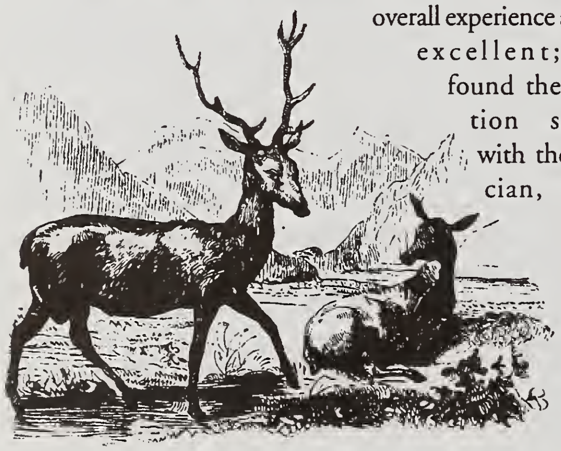
physician's office. DEARS participants are asked to fast for 8-12 hours prior to arriving at the DEARS site, and to bring all their prescription and over-the-counter medications. The program starts at 7:30 A.M., when participants are checked-in and blood and urine samples are obtained for laboratory tests that the physician had ordered several days earlier. These specimens are sent immediately to a laboratory that provides results within two hours of receipt. Afterwards, participants are provided breakfast courtesy of the Diabetes Control Program. After breakfast participants take part in group education sessions with the nurse, pharmacist, dietitian (all three are certified Diabetes Outpatient Educators) and physician. At these sessions, members of the team can deliver and reinforce standardized diabetes self-management information, and help participants practice diabetes self-management techniques. Group education sessions are interwoven with one-on-one sessions with the nurse, dietician, and pharmacist. The pharmacist reviews participants' medications to ensure that participants are taking prescription medications as ordered and to detect any interactions. Lastly, each participant meets individually with the physician to review results of his/her laboratory tests. The physician also makes referrals for additional dilated eye and podiatry examinations and formal diabetes management education as needed.

Before leaving, patients schedule additional visits with the physician, as appropriate, and are given a participant satisfaction survey. Individual members of the DEARS team are also queried on their experience with the program, to pinpoint possible deficiencies in the program. The session ends at noon.

To date, three DEARS sessions have been conducted with the full support and participation of physicians in three different practice settings - primary care, specialty care, and a senior center. Each session has served approximately 10 DEARS participants, and the physicians have billed Medicare for level 2-3 visits. Physician-participants have commented favorably on the patient flow, the usefulness of the medical abstraction form, the group education sessions, and the team approach. In addition, one noted that it was "...gratifying to get as much assistance in educating my patients." Participating physicians have agreed to serve as mentors for other physicians wishing to incorporate DEARS into their practice.

Patient-participants have also commented favorably on DEARS. Of the 30 participants to date, 100% rated their overall experience as good/excellent; 100% found the education sessions with the physician, nurse, pharmacist, and dietician somewhat/very useful; 100% liked the group education format and meeting the diabetes team that day somewhat/a lot; 96% liked getting their test results back that day somewhat/a lot; 93% liked meeting others with diabetes somewhat/a lot; 89% liked reviewing their medications with the pharmacist somewhat/a lot; 93% would probably/definitely return for a DEARS program next year; 93% would probably/definitely change the amount of food they consumed; 90% would probably/definitely change their self-monitoring of blood sugar; 86% would probably/definitely changes the kinds of food they eat; 72% would probably/definitely change the kind of physical activity they performed; 71% would probably/definitely change the amount of physical activity they performed; and 48% would probably/definitely change their medications.



The DEARS approach to the management of diabetes standardizes much of the information that patients need to manage their diabetes and to modify their risks for complications. It efficiently and cost-effectively uses scarce resources, physicians' time in particular, while maintaining physician reimbursement. It improves patient access to comprehensive diabetes care ("one-stop-shopping"). The program is portable and flexible enough to adapt to different types of medical practices without disrupting office procedures and patient flow. It is provided by the Rhode Island Department of Health at no cost to participating physicians, and as often as physicians request it. Perhaps most importantly, for physicians who are increasingly concerned that managed care constrains the quality of care, the DEARS program offers an easy way to integrate numerous quality assurance measures imposed by Medicare and managed care into their medical practices. For physicians and patients alike, the DEARS program builds networks for the achievement of a common goal - better adherence to diabetes standards of care, hence longer lives and better quality of life for persons with diabetes.

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A Novel Diabetes Program

Raymond Maxim, MD

The last time I addressed this column I briefly discussed the DEARS program (Diabetes Education, Assessment, Referral and Screening). You will find a more detailed description in this issue of *Medicine & Health/Rhode Island.*[1] However, I would like to take some time to give a personal account of my experience with this exceptional program.

Last July, I was asked to participate in the program. At that time, I was struggling to coordinate the care of my diabetic patients. I was finding it difficult to manage the appropriate referrals. Dietary counseling, for example, was frequently delayed, so the teaching that patients received in my office was not reinforced in a timely fashion. As most of my diabetic patients are elderly and many no longer drive, transportation to the dietician, ophthalmologist or podiatrist can be difficult. In addition, diabetic teaching and self-glucose monitoring education was taking a great deal of my office staff's time and energy. I felt compelled to take advantage of this opportunity.

A week prior to the arrival of the DEARS team, my office staff was asked to pull the records of ten diabetic patients. I decided to get the most benefit out of the program and picked ten of my patients having the most difficulty controlling their diabetes. Then the DEARS team sent out a member of the team to review the records for needed services. If laboratory testing was needed the appropriate lab slips were completed. If there was no evidence of a foot exam or dilated funduscopic exam in the patient chart this was noted on the DEARS clinical summary form (this record was available for me to review during the individual exam and counseling session). The team and I also evaluated the degree of blood pressure control as well as the appropriateness of the antihypertensive agents and ACE inhibitors and beta-blockers if they seemed suitable. Patients arrived at my office fasting, had the missing or scheduled laboratory work done, and then were given a complimentary breakfast. Later, patients participated in an interactive educational session, the details of which can be found in the accompanying article. During the morning, there is an opportunity to address the group as a whole. I highly recommend that you take advantage of this occasion. It reaffirms to your patients that you care and it gives you a greater awareness of how well your patients understand their disease.

One physician received a standing ovation from his patients when he entered the room after the session.

What are the advantages to the physician who invites the program into their office?

1. A diabetic patient population with a better understanding of their disease process.
2. A diabetic patient population likely to be more compliant with your instructions.
3. You or your staff need less time to gather needed laboratory data.
4. Fewer screening tests are missed or delayed.
5. Less staff time is needed to provide diabetic teaching.
6. The teaching time that you or your staff provides will reach a better educated and more motivated patient.
7. A better rapport with your diabetic patients. One physician received a standing ovation from his patients when he entered the room after the session.
8. Reduced liability for not meeting the standards of care in a recalcitrant patient population.
9. You are able to bill for your individual patient encounter.
10. Provide the documentation necessary for billing a more complex visit.

Overall, I found the DEARS program to be an enjoyable and effective process. The majority of my patients who participated in the program have made significant strides in control of their disease. As a result, I have had many requests to repeat the program from previous participants and from patients who heard about the program from the participant. I highly recommend that you give the program an opportunity to enter your office. It is very flexible and can fit just about any practice environment.

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Raymond B. Maxim, MD, is Associate Clinical Coordinator, RIQP; Clinical Instructor, Brown University School of Medicine; and staff physician, Roger Williams Medical Center.

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The author assumes full responsibility for the accuracy and completeness of the ideas presented. This article is a direct result of the Health Care Quality Improvement Program initiated by the Health Care Financing Administration, which has encouraged identification of quality improvement projects derived from analysis of patterns of care, and therefore required no special funding on the part of this Contractor. Ideas and contributions to the author concerning experience in engaging with issues presented are welcomed.

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Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	Nov. 1998	12 Months Ending with Nov. 1998		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	259	3,118	314.9	3,683.5
Malignant Neoplasms	201	2,473	249.7	6,598.0**
Cerebrovascular Diseases	42	599	60.5	783.5
Injuries (Accident/Suicide/Homicide)	19	351	35.4	6,630.5
COPD	30	453	45.7	345.0

Vital Events	Reporting Period		
	May 1999	12 Months Ending with May 1999	
	Number	Number	Rates
Live Births	1,185	13,582	13.7*
Deaths	736	9,699	9.8*
Infant Deaths	(7)	(91)	6.7#
Neonatal deaths	(6)	(69)	5.1#
Marriages	824	7,500	7.6*
Divorces	196	3,147	3.2*
Induced Terminations	339	4,802	353.3#
Spontaneous Fetal Deaths	36	1,059	78.0#
Under 20 weeks gestation	(31)	(981)	72.2#
20+ weeks gestation	(5)	(78)	5.7#

**Excludes one death of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 990,225

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

* Rates per 1,000 estimated population
# Rates per 1,000 live births

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Ninety Years Ago

[November, 1909]

D.L. Richardson, MD, in "The Value of the X-Ray in the Treatment of Psoriasis," recounted his experiences as a patient and a clinician, citing 6 cases. Along with x-ray treatment, he advised continuing conventional treatment ("omission or reduction in use of meat, relief of constipation by proper medication, and the use of arsenic, sodium salicylate or thyroid extract"). As for the reason x-ray therapy worked, Dr. Richardson conceded, "I think that is impossible to be explained with certainty."

At a meeting of the Providence Medical Society (69 members present), Dr. F. E. Peckham concurred with the importance of x-rays: "In...fractures or injuries to the neck and head of the radius there is no earthly way of knowing the real trouble without an x-ray picture."

At that same meeting, Dr. Bowen discussed abortions: "...Hat pins seem to be the favorite home remedy. I was called not long since to see a woman who was losing flesh...She had an abortion and I found out afterward that she had been using a hat pin" [Another patient) had used the tip of a fountain pen. "Her efforts were unsuccessful. She had bumped down several stairs without result...She jumped off the sewing machine, which did the work. This patient stayed in bed six weeks." Dr. Dyer cited a 45-year old woman: "She told me she had 22 abortions in her life, 2 of which had been performed by an an obstetrician, and the other 20 by herself. She had a wire that she used and she was very careful of it."

Fifty Years Ago

[November, 1949]

Walter C. Alvarez, MD, from the Mayo Clinic, described "Some Common Syndromes Often Misdiagnosed" at the 138th meeting of the Rhode Island Medical Society. He traced misdiagnoses to hurried physicians: "Usually, the doctor felt he was too busy to take an adequate history.... He tried to practice 'decerebrate medicine." For example, a doctor might diagnose "dyspnea" in a woman who can play tennis vigorously during the day, but wakes at night "feeling she can't get air down." "Perhaps before she went to sleep she had a row with a daughter or husband." Her hyperventiliation led to dizziness, which led the family to call a doctor. "If on arrival he recognizes air hunger, if he reassures the woman, gives her a little bromide and sends her back to bed, all is well. But if he rushes her to a hospital and gets a dozen electrocardiograms and finally says that she has had a coronary attack she may be a cardiac neurotic for the rest of her life."

Frederick A. Webster, MD, in "Double-Kidneys," reported on two cases, both mistreated for years - one as an orthopedic problem, the other as a psychoneurotic complaint.

Edward J. Heitzman, MD, and Frank B. Cutts, MD, in "Sub Acute Bacterial Endocarditis," reviewed 45 cases and found 9 patients who had had teeth extracted. The authors called "...recent dental extractions without the prophylactic use of of penicillin" an important cause.

Twenty Five Years Ago

[November, 1974]

In "Message from the Dean," Stanley M. Aronson, MD, reported on Rhode Island students' rate of admission to medical schools. In 1973 and 1974, 30.7% and 32.3% of Rhode Island applicants were admitted to medical schools (national rates were 34.6% and 32.1%). The acceptance rate per 1 million population for Rhode Island was lower than the national rate (in 1974, 42.1 versus 62.8), largely because there were fewer aplications. Rhode Island did better than students nationally on all parts of the MCATs.

David Morris, DPhil, Chief of Biochemistry Department, The Miriam Hospital, discussed "Drug Metabolism and Therapeutic Monitoring: New Technical Procedures Permitting Accurate Assays will Play Important Role in Improving Patient Care." The analytic chromatographic procedures for quantification and identification of drugs - initially used in the diagnosis and treatment of overdoses - were "increasingly used to monitor individuals for levels of therapeutic drugs."

Banice M. Webber, MD, and Arvin S. Glicksman, MD, in "Radiation Therapy in the Management of Patients with Breast Cancer," advocated radiation for low and regional recurrences following mastectomy.

Michael Ingall, MD, discussed "Psychiatric Casualties of the Exorcist." After the film opened in February 1974, 6 new patients at the Providence Mental Health Center complained of possession by the devil.

Albert S. Most, MD, and Robert J. Capone, MD, praised "The Echoccardiogram" as "... an important diagnostic technique in cardiac disease."

An editorial on obesity ("Too Many Eat Too Much") blamed eating patterns established early in infancy and childhood.

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Cover: "59 Ford," by Steven Boksenbaum, a Pittsburgh artist long interested in mid-century diners and automobiles. His work is slated for display in the forthcoming Historical Diner Museum in Providence.

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COMMENTARIES

Driving and the Physician: A (More) Modest Proposal

Assessing a patient's ability to drive is a contentious problem for physicians. We have no training to assess driving ability and the evaluation frequently puts the doctor in an adversarial role. The extremely interesting and educational articles in this issue make clear both how much is known about driving safety in medically affected and older populations and how much more there is to learn. I think it is perhaps a shock to the physician-parent to learn that the only population at greater risk of motor vehicle accidents than the demented elderly is the male teenager.

There are two types of medical problems that interfere with driving, those that are episodic and those that are permanent. The former includes epilepsy, diabetes, cardiac arrhythmias, sleep disorders, and behavior disorders, among others. The latter are blindness and the host of neurological, psychiatric and drug-induced impairments that affect judgment and reaction time. Each type of problem is hard to assess, but criteria for allowable limits for episodic behavioral abnormalities can be developed, using the number of episodes modified by the duration and quality of symptoms allowed per unit of time. The permanent neurological impairments from stroke, Alzheimer's and Parkinson's diseases cause a wide spectrum of problems and there are no clear-cut criteria for restricting licenses. In the few studies published on driving safety in Parkinson's disease one discovers that accidents correlate with dementia rather than motor impairment. Presumably this means that PD patients with insight voluntarily restrict their driving, and limit themselves to the safest conditions consonant with their disability. The demented lack insight and fail to do this.

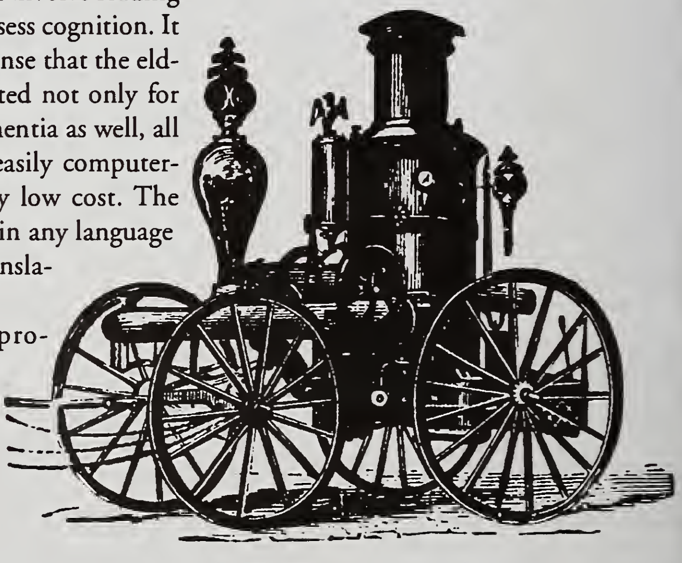
We learn in this issue that demented drivers do worst in unpredictable, sudden situations. Yet these situations are not part of the standard road test, and would be difficult to incorporate. Currently the only test required for maintaining one's license is the vision test.

I think there is universal agreement that many demented and other impaired patients should not be driving. There is disagreement only over how severe the impairment must be. Computerized tests have been developed which do not involve reading and can reliably assess cognition. It therefore makes sense that the elderly should be tested not only for vision but for dementia as well, all of which can be easily computerized at a relatively low cost. The tests can be given in any language without using translators.

My simple proposal is that all drivers over the age of 70 be tested every two to five years (frequency to be determined) and that vision (day and night), rules of the road, and cognition should be assessed. Drivers with conditions that may affect driving safety who require safety checks should be referred by their doctors, regardless of their age. Those who fail a screening test must then pass a road test. Funding for this should come from the insurance companies, which would save significant sums by having unsafe drivers removed from the roads.

I also recommend the development of restricted licenses for people with mild impairments.

– Joseph H. Friedman, MD



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The Physician Chekhov Inspects a Russian Prison Camp

Anton Chekhov had been practicing medicine in Moscow for six years, augmenting his meager income by writing short pieces of fiction for the popular magazines of his day. The four great plays which would insure his literary immortality as Russia's foremost dramatist had yet to be completed; but still his prominence as a author had already surpassed his renown as a medical practitioner.

The year was 1890 and Chekhov was experiencing a growing dissatisfaction with the modesty of his medical skills. He pondered frequently on how little he had learned since graduating from Moscow School of Medicine in 1884.

Yet another problem now loomed. Chekhov knew that he suffered from tuberculosis but thought little of its consequences until his beloved brother Nicholas, a gifted artist, suddenly died of the disease.

Chekhov's degree in medicine gave him license to practice; but a doctorate in medicine, which he did not possess, required an additional year of disciplined research culminating in a written thesis acceptable to the senior medical faculty. Chekhov's feelings of professional inadequacy and the ominous message conveyed by his brother's premature death forced him now to reflect upon his career and how his remaining professional years might be most effectively employed.

Instead of continuing his meager practice he elected instead to undertake an epidemiologic study of the medical problems in Russia's extensive prison facilities; his specific locus of inquiry, much to his family's dismay, was to be the remote and foresaken Siberian island of Sakhalin, a territory annexed by Russia in 1875 and used primarily for the exile of its more dangerous and intransigent prisoners. Russia, of course, had been regularly dispatching its unwanted to Siberia since 1550 long before any Gulag system had been established. Siberia's vast wilderness was punctuated by a series of prison settlements populated by felons, political dissidents and social outcasts. Sakhalin was the furthest removed and the most forbidding of these settlements.

Sakhalin was an island some 600 miles in length and originally peopled by aboriginal tribes. Russia intended to colonize this inhospitable territory with prisoners who, after completion of their sentences, would be encouraged to remain. Each year, many thousands of Russians, some accompanied by their wives, were shipped east across the vastness of Siberia to this snow-covered, fog-enshrouded Pacific island north of Japan. Few returned to European Russia, most dying of malnutrition, the harsh winters or intercurrent disease.

Before embarking upon on this investigative venture, Chekhov studied available texts concerning the ecology of the island, its meager resources, its history, the barbaric penal code of imperial Russia, the demography of the prisoner population and whatever archival reports had been submitted by the governors of Sakhalin.

Chekhov's perilous journey began in April, 1890, with the crossing of the Ural mountains and the traversing of Siberia using primitive carriages and river ferries. [The trans-Siberian railroad was not yet begun.] The hazardous trek to the port of Nicholaev at the mouth of the Amur river covered 5,000 miles and occupied almost four months. A steamer then transported him to Sakhalin island.

Russia at this time was undergoing much political unrest; and many of its more articulate intelligentsia - writers, physicians, teachers - were committed to the downfall of its tsarist regime. Chekhov, however, considered himself apolitical and removed from the debate between those who viewed the Russians as eternal children needing strong paternal authority and those moved more by Western ideas of democracy. Little in his writings even acknowledged the existence of an oppressive, authoritarian regime. Chekhov was not therefore viewed as a threat to the government and was therefore accorded wide latitude to interview and examine the prisoners.

Chekhov worked feverishly for four months conversing with many of the resident prisoners and former prisoners. Without complaint he endured the island's squalor, battalions of cockroaches, wretched weather and atrocious living conditions. And during this interval of intense field investigation, he accumulated written records on over 10,000 men and women living on Sakhalin island.

Satisfied with his studies, Chekhov packed his belongings and on October 13, 1989, returned to Moscow by steamer with stops at Vladivostok, Hong Kong and Ceylon. After passage through the recently constructed Suez Canal he disembarked at the Black Sea port of Odessa.

In the year that followed his return to Moscow Chekhov was preoccupied with an outbreak of cholera superimposed upon a regional famine. And because of these unanticipated demands on his time, the complete Sakhalin text was not published until 1894.

The final report turned out to be more than a mere travel journal or personal memoir. In dispassionate, nonpolemical language, Chekhov provided the readership with an exhaustive analysis of the archaic Russian penal code, its structural inhumanities and its corrupt operations. His text described a capricious system which detained large numbers of Russians, many for political reasons, and through the perverse alchemy of its punitive methods either killed them or converted them into incorrigible criminals. His report was a damning document and moved the tsarist government in the direction of penal law reform.

And Anton Chekhov's subsequent life ? Shortly after his return from his stressful mission to Sakhalin, his tuberculosis became active eventually requiring that he retire to the warmer climate of the Crimea. He died of tuberculosis at age 44, 14 years after the Sakhalin venture. These were not idle years however. Chekhov's greatest dramas were written during this interlude.

And the fate of his Sakhalin report? It went on to become a sentinel document encouraging many western nations to review their penal procedures. It was also duly submitted to Moscow University's dean of medicine as Chekhov's doctorate thesis. The dean summarily dismissed it as unscientific trash.

– Stanley M. Aronson, MD

Driving Policy Issues and the Physician

Brian R. Ott, MD, and Stephen T. Mernoff, MD

This issue of *Medicine & Health/Rhode Island* provides us with important and practical information about how medical illness can impact driving ability, which hopefully will assist physicians in counseling their patients. The specific problems presented include age-related impairments and medical illness, dementia, epilepsy, and other disorders of consciousness. New approaches to cognitive and functional assessment of medically impaired drivers are also discussed.

Strictly speaking, driving is a privilege granted by the state based upon age restrictions as well as the ability to pass a vision test, written examination concerning knowledge of driving rules, and practical road test. The driving privilege ultimately can only be revoked by the state.

Despite never having had formal training in the evaluation of driving ability and a relative dearth of published research in the area, physicians are often called upon by the state to render opinions about a person's fitness to drive. Several studies have shown that physicians are often unaware of the applicable legal requirements and protections in the states in which they practice.[1,2]

Furthermore, the physician is left in the uncomfortable ethical position of being a patient-advocate who maintains confidentiality in delivery of care, yet is called upon by society to report patients suspected to be incompetent drivers for the sake of public safety. Thus the physician has a moral, though poorly defined and not necessarily legal, obligation to the public at large to report. Development of clearly defined public policies in this area should reduce the physician's dilemma by providing appropriate guidelines.

WHAT ARE THE PRESENT PUBLIC POLICIES?

A major difficulty of applying risk data to driving policy is the issue of how to determine the "acceptable" degree of risk. Should the goal be to reduce the risk to that of the general population, or would a higher risk be "acceptable" to allow selected people to have access to the economic and social advantages of driving? Who should determine the "acceptable" risk:benefit ratio? Using what criteria? Society has placed this burden on Departments of Motor Vehicles (DMV), which often then place responsibility on physicians. Clearly so many factors are involved that the decisions must be individualized for each patient. The data can only provide general guidelines which must be neither too general nor too specific.

Abbreviations Used:

DMV	Department of Motor Vehicles
ICD	implantable cardiac defibrillators
MAB	Medical Advisory Board

In Rhode Island drivers are required to renew their licenses every two years instead of every five after age 69.

DEMENTIA AND AGING

At present California is the only state which specifically mandates reporting of demented drivers. Six other states have statutes with mandatory reporting of medical conditions that impair driving.

In Rhode Island drivers are required to renew their licenses every two years instead of every five after age 69. The renewal process involves a test of vision, but no tests of knowledge, cognitive ability, or driving competence. Concerns about physical and mental impairments may be brought to the attention of the DMV Medical Advisory Board through multiple avenues including self-report on the license application, observation by the licensing examiner, reports from police officers, family or physician, or a personal driving record of crashes and/or violations. This leads to a medical evaluation form that is completed by the physician. A driving test may be requested. Although a special restricted license is one option for the driver with mild cognitive impairment, in actual practice the reported driver ultimately either keeps his license with full privileges, or has it suspended.[3]

Once a person is found to be incompetent, the issue of advising that person to abstain from driving is particularly problematic since many persons with dementia do not recognize their deficits, or if they do, they tend to minimize their significance and not comply. The issue of medicolegal liability for physicians who do not advise demented patients to abstain is not clear. Court decisions have been divided on this issue when applied to cases of persons with other neurological disorders such as stroke and epilepsy.

The most serious threat to the elderly individual faced with the prospect of losing his driving privilege is the loss of independence that may result. Driving may be the only way for an elder to maintain social contact or provide necessary transportation for self and spouse.[4] This dignity and quality of life factor needs to be seriously addressed in any public policy or individual decision to revoke driving privileges. Driving restriction should not be imposed prematurely. The reasons for termination of driving need to be discussed openly with the person and alternative forms of transportation be suggested or provided.

Compromises such as limited driving parameters or a graduated program of driving reduction can be made, at least in the early stages of disease when some degree of insight is preserved. "Low-risk" privileges may include short trips, slow roads, daytime driving, and non-rush hour driving. The addition of a copilot will help to avoid problems of getting lost, running out of gasoline, or arranging for emergency repairs.[5]

The use of sedative medications has been reported as a risk factor for crashes among elderly and demented patients. Attention to minimizing the use of such medications and the elimination of alcohol, as well as maximizing function by treating underlying medical conditions, visual, and hearing deficits is advisable. To assist elderly drivers in evaluating and improving their skills there are several programs in the area including those sponsored by the American Association of Retired Persons, the American Automobile Association, and the National Safety Council.[3]

Forced restraint from driving may be required for those dementia patients with more advanced disease who do not comply with voluntary restriction.[4] Unfortunately revocation of license does not necessarily lead to driving cessation, particularly when the patient lacks insight and judgment. As a last resort the keys can be taken away or the vehicle disabled by removing the distributor cap or the wire leading to it. The vehicle can be removed to a distant site, e.g., for "repairs." If the vehicle is sold, the money earned from the sale as well as money saved from insurance can be used to pay for public transportation.[6]

EPILEPSY AND DISORDERS OF CONSCIOUSNESS

The vast majority of states and provinces have driving policies regarding epilepsy, stroke, cardiovascular disease, and diabetes. The policies tend to be vague and variable,[7] although this is beginning to change. Table 1 shows the distribution of seizure-free intervals recommended (as of 1994; adapted from Krumholz[8]). [Data from 1994 are the most recently compiled.]

In general, driving policies in the U.S., for all medical problems, are less restrictive than in other countries, but vary considerably from state to state as far as the details. Some provide for restricted licenses (e.g. driving for work or food only). Others make provisions for specific circumstances (e.g. if seizure recurrence is due to a known, reversible acute cause in an otherwise well-controlled individual the seizure-free interval requirement may be waived). Most, if not all, states need to offer policies with greater detail and flexibility.[9]

Table 1.
Driving and epilepsy policies in the U.S.

Seizure-free interval (months)	# of states*
3	6
6	15
12	22
18	1§
24	1
none	7

* 50 plus Washington, DC,. and Puerto Rico
§ Rhode Island, as of 1994, see text

Most states have mechanisms of administrative review with the input of the treating physician and, if necessary, a medical advisory board. Physicians do not make the final decision, which rests solely with the appropriate state agency. According to a 1992 report[8] of 63 states and Canadian provinces, physician reporting was mandatory in 12, immunity laws were in place in 36, and physician reports were confidential by statute in at least 44. A discussion of the pros and cons of immunity laws and mandatory physician reporting is beyond the scope of this paper.

Wisconsin was the first state to permit driving by those with well-controlled seizures. Over the years, Wisconsin found that liberalization improved driver compliance with policies and mishap rates were comparable to non-epileptics.[7] As of 1997, Wisconsin recommended a 3-month seizure-free period based on data suggesting that the predictive power of 3 event-free-months is about the same as 6 event-free months.[2]

At this time, there are no formal statutes or DMV regulations in Rhode Island regarding driving for individuals with seizure disorders, syncope, or other specific medical conditions. There are some written policies in archaic language regarding "habitual drunkards" and "habitual users of . . . drugs" and those suffering from "physical or mental disability."[11] These are general and simply give authority to the DMV to withhold licenses from those the Registry believes are not competent to drive safely. According to Mr. Philip Lagoy, Hearing Officer Liaison for the Medical Advisory Board, the DMV is currently developing formal, written rules and regulations for various medical problems, including habitual alcohol use and seizure disorders.

The unwritten guideline for seizure-free interval has traditionally been 18 months; however, recently Dr. John Strom of the Medical Advisory Board (MAB) has emphasized that there are no rigid requirements. All cases of epilepsy, syncope and loss of consciousness that are referred to the DMV are individually reviewed by the MAB. Information comes from accident reports, physicians, family, medical forms, consultation reports and other sources; the DMV may provide driving records that are medically pertinent. Neurological evaluation and an EEG are required for all cases of new onset epilepsy and isolated seizures, as well as for cases with incomplete information.

The recommendation for patients with epilepsy is based on the etiology, type and pattern of seizures, age, personal and medical history, and the circumstances of the most recent seizure. In general, the required seizure-free interval for uncomplicated new onset, isolated or recurrent seizures is three to six months. This is not fixed, however, and may be extended up to twelve months, while for other cases there may be no waiting period.

Currently there are also no specific written guidelines in Rhode Island regarding cardiac or sleep disorders. In the US, as of 1991, 26 states recommended no driving for anywhere from 3-12 months after an episode of non-seizure syncope; only eight states restricted driving specifically for dysrhythmias for between 1.5 and 18 months. In Rhode Island there is no written policy regarding arrhythmias, but this is currently being reassessed. In Massachusetts, as of June 1998, the policy[12] for noncommercial drivers with implantable cardiac defibrillators (ICD) is similar to the seizure policy: a six-month event-free period and detailed physician statement are required after ICD implantation or after an ICD trigger episode for a license to be reinstated.

Reporting Policies and Procedures in Rhode Island

In Rhode Island reporting of medical conditions to the DMV is not mandatory. Physicians who do choose to report a patient in good faith are protected from litigation by the reportee and confidentiality of the report is preserved. Nine other states provide similar statutory authorization for reporting.[3]

Rhode Island physicians choosing to report a hazardous driver should write to Mr. Allen Simpkins, Administrative Adjudication Court, Office of Operator Control, 345 Harris Avenue, Providence, RI 02909. The letter may contain a recommendation for license suspension based on the person's medical status, in which case the license would be suspended. For cases in which the physician is in doubt regarding the patient's driving competence, the letter may contain a request that a practical road test be performed.

The DMV uses information supplied by physicians to make its decision; questionable cases are referred to the MAB, which meets monthly. The MAB is a review/advisory panel, and the DMV makes the final decision. Members are appointed for 3-year terms and must include at least one of each: general practitioner, neurologist, psychiatrist, optometrist, orthopedist, physician, and two members of the public (one each representing the elderly and the disabled). The current MAB rules and regulations refer to medical issues in general terms only: "The standards applied. . . shall be consistent with developments in the medical field that relate to . . . fitness to operate a motor vehicle and. . .shall recognize the technological advances in the rehabilitative field which may enable a handicapped person to safely operate a motor vehicle. . ..."[11]

The driver has the right to review the physician's letter and any other materials in his/her file. Once the Division of Operator Control receives the letter, it will notify the person of a license suspension or will request that the driver appear for an informal hearing. The hearing officer will review the information and may request that the person take a road test either before or after the hearing. The hearing officer also may request additional medical information from the driver's physician. If the driver fails to appear for the hearing, the license is automatically suspended. If the hearing officer orders the driver's license suspended or revoked after the informal hearing, the driver has a right to appear before the MAB with legal counsel to appeal the decision.

Future Directions

Further research needs to be done to define valid risk factors and reliable office screening procedures for physicians to use in identifying those most at risk. In the case of dementia, for example, determination of cutoff scores on mental status tests or measures of instrumental activities of daily living would be useful that could predict a 95-100% likelihood that a driver would pass or fail a road test. Development of valid driving simulation tests could replace actual road testing of those identified as at-risk drivers. While at the present time road testing may be regarded as the "gold standard,"[13] such testing does not clearly predict performance in stress situations, and can be both time-consuming and expensive to institute on a large-scale basis.

Efforts are underway among medical experts in driving research to develop consensus of opinion regarding medical disorders and driving. In 1994 a work group of driving and dementia experts was convened by the Swedish National Road Administration. A consensus statement from this group recommended that individuals with moderate to severe dementia be advised not to drive; however, there was no agreement on appropriate cutoff scores for standard mental status screening measures such as the Mini-Mental State Examination. Those with a "mild" degree of dementia may also be at risk and should be considered for specialized assessment of their driving competence.[14] The American Psychiatric Association Practice Guideline makes similar recommendations.[15] The American Academy of Neurology is currently developing a Practice Parameter for dementia and driving.

Also in 1994, the American Academy of Neurology, the American Epilepsy Society and the Epilepsy Foundation of America endorsed consensus statements regarding driver licensing and epilepsy.[16] They provide sample statutory provisions regulations. The authors of these guidelines used both data and the experience of several states (particularly Wisconsin) to provide sound recommendations to states as they update their vague and obsolete policies.

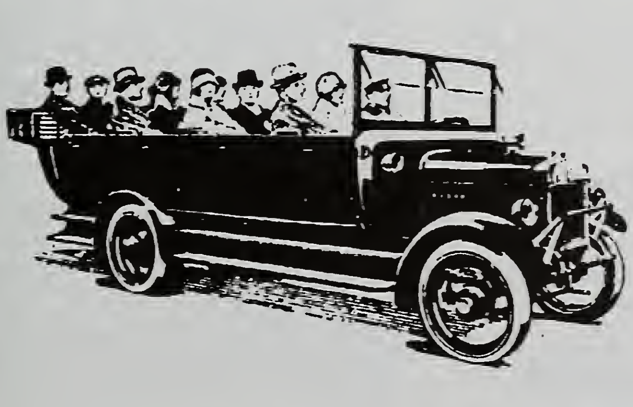
Key points of this statement regarding physician involvement in the decision are summarized below. Recommendations specific to those with seizure disorders are summarized as follows:

- Seizure-free interval: "3 months is preferred. . . modifiers could alter the interval."
- Unfavorable modifiers (selected):
  - Noncompliance with medications/office visits
  - Poor driving record
  - Alcohol/drug use
  - Crashes due to seizures in previous 5 years
  - Increased seizure frequency in past year
- Favorable modifiers (selected):
  - consistent prolonged auras
  - pure nocturnal pattern
  - seizures due to other unlikely-to-recur condition lowering seizure threshold (acute illness, sleep deprivation)
  - simple partial seizures that do not interfere with motor control

Although written with epilepsy in mind, this consensus statement provides other guidelines that are broadly applicable to a range of medical disorders that could affect driving competence.[16]

- The state DMV, not the treating physician, should make the licensing decision. Physicians should complete a form describing the pertinent medical facts; the physician should have the option to make an opinion on whether or not the individual should be licensed, but such an opinion is not mandatory.
- First-level DMV staff should be able to make the licensing decision on the basis of these forms in most cases.
- Patients should be required to report the seizure disorder to the DMV and report recurrent seizures; the physician should have the right to report, with immunity, a patient who has not reported himself and is putting the public at risk.
- Physicians should have immunity for choosing both to report and to not report a patient to the DMV if their best judgment is used; such a report should not be allowable in other legal proceedings.
- Licensing criteria for various conditions should be in regulation and guideline form rather than by statute; criteria should be developed by a group with expertise in that condition.
- Criteria should be detailed but flexible enough to allow case-by-.case review if necessary.
- "...physicians should not be required to report their patents to the DMV" but should discuss risk issues with patients and tell them about the DMV requirements (including self-reporting obligation).
- A medical advisory board should exist to set and periodically review medical standards for licensing, educate physicians and the public, and review complex cases.
- A mechanism for a hearing and an appeals process should be in place.

Finally, any broad public policy must take into account the need for alternative transportation for anyone who loses a license for medical reasons. A more widely accessible and affordable system of public transportation as an alternative to driving needs to be developed. Discussions in public forums as well as among government, healthcare, insurance and automobile industries will be needed as we grapple with these problems in years ahead.



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The Elderly Driver

Mark I. Schwager, MD

The number of elderly persons who drive continues to grow, and an ever increasing percentage of elderly people have licenses.[1] The age of drivers reflects the age demographics of our population at large. The population aged 65 and over is now growing more rapidly than any other age group. An acceleration of the growth of the older population will begin in 2010 when the baby boomers become part of the over 65 population. By 2020, 17% of the US population will be over 65, resulting in over 50 million elderly persons eligible to drive.[2] Social changes will also increase the average age of drivers. The baby boom generation of women drivers will result in a substantial increase in the number of elderly women who drive in the next century. Improved functional status of the elderly will also swell the ranks of our senior drivers as Americans aged 65 today can expect to spend at least 2/3 of their remaining 15-year lifespan living independently. The elderly population has also become increasingly suburban as it "ages in place." This increases the dependency on the auto and keeps elderly drivers on the road and increases the number of miles driven per year.[3] It is unlikely that other modes of transportation can provide anywhere near the level of mobility nor user-friendliness that the elderly want or need.

Changing Physiology and Health of the Older Driver

Even in healthy aging there are dramatic changes in metabolism, thermoregulation, immune response, cardiorespiratory endurance, muscle strength, neurologic function, vision and hearing. Chronic medical illness increases in prevalence with increasing age. Of those older than 65, over 80% have at least one chronic disease; the majority have two or more. Cognitive impairment increases with age, with rates for Alzheimer's disease approaching 40-50% after age 85. Thus, as the age of the driving public increases so will the representation of people on the road with a variety of chronic medical conditions. We will increasingly be involved in counseling patients, families and public officials on limiting or terminating driving privileges in the elderly. Presently, physicians lack a standardized tool to evaluate driving risk in seniors and public policy makers have not decided where and how to implement procedures to identify impaired drivers. There is, however, a growing amount of information to help clinicians make reasonable decisions.

Abbreviations Used:

AARP	American Association of Retired Persons
UFOV	useful field of view

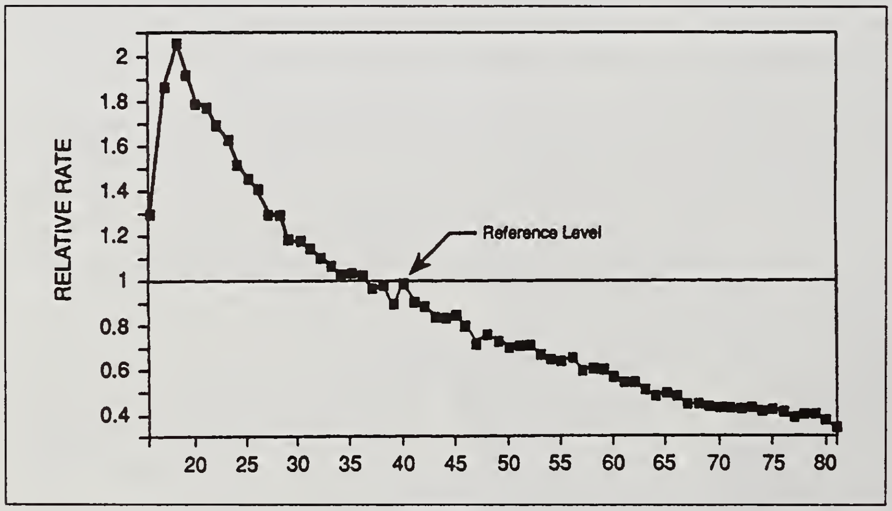

Figure 1. Rate of motor vehicle crashes according to age of driver.
SOURCE: Cerrelli E. *Older Drivers: The Age Factor in Traffic Safety.* Washington, DC: US Department of Transportation, National Highway Traffic Safety Administration, 1989

Risk of Motor Vehicle Crashes

Elderly drivers account for a lower proportion of crashes than any other age group (Figure 1), and the absolute number of crashes involving the elderly is low. The average road-user is at much greater risk of injury by a healthy, younger driver with poor judgment. Nevertheless, elderly drivers do have high crash risk when adjusted for miles driven. Accident rates per mile driven increase steadily beyond age 60,[4] although the increase is not very marked before age 75 (Figure 2). An average 65 year-old driver travels approximately 6800 miles per year while an average 80 year-old drives about 2500. Thus, the older driver is on the road less so that adjusted for mileage, the number of crashes per mile is high.

There is also a greater likelihood of injury, hospitalization and death among older individuals following a crash, making motor vehicle crashes the leading cause of death from unintentional injury for those aged 65 to 74. Crashes are about 3.5 times more likely to be fatal for older drivers than drivers under 65 years old. Elderly drivers also incur greater physical injuries than younger drivers involved in similar accidents.[5]

Patterns of Crashes in the Elderly

While younger groups of drivers have proportionately more single vehicle crashes, drivers over age 60 and especially over 75 have more right angle

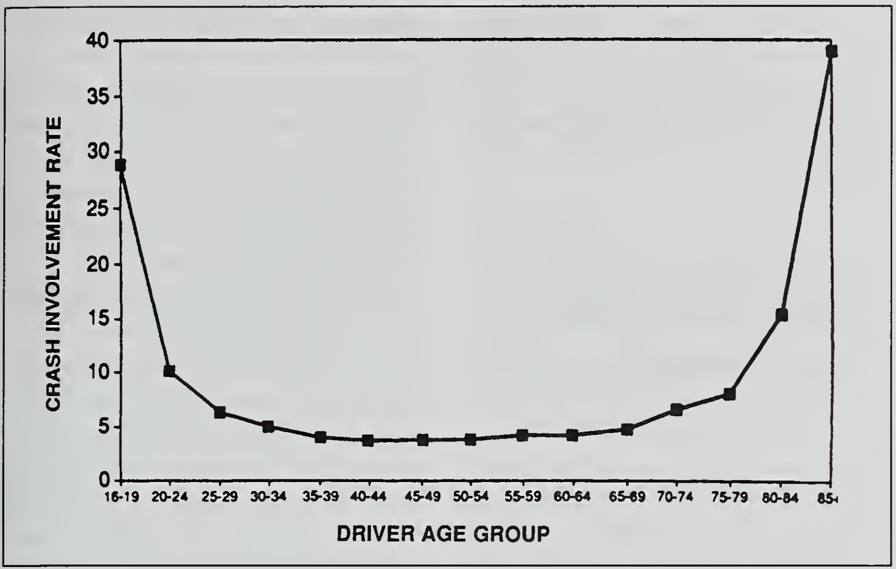

Figure 2. Rate of Motor vehicle crashes adjusted for miles driven according to driver's age. SOURCE: Cerrelli E. *Older Drivers: The Age Factor in Traffic Safety.* Washington, DC: US Department of Transportation, National Highway Traffic Safety Administration, 1989

crashes and crashes associated with rights of way, intersections and left hand turns.[6] Left hand turns are particularly problematic, exposing drivers to three lines of traffic. Older drivers are adequate with automatic processes which place limited demands on attentional capacity and generally occur under highly predictable conditions of traffic and weather. Non-automatic processes are those used to deal with unpredictable or unfamiliar demands such as turning, merging, collision avoidance or driving in adverse weather conditions. These non-automatic processes are more challenging for older drivers. Of interest is that the standard licensing road test assesses mostly automatic processes and will miss impaired ability to react appropriately to unpredictable, or complex situations.

Crashes in the Elderly: A Geriatric Syndrome

Studies on the impact of specific medical diagnoses on driving have been conflicting and there is no agreement across studies on which medical conditions elevate crash risk.[7] Diagnosis alone is insufficient to determine driver competence. There are those within any diagnostic category with competent driving skills who are little risk to traffic safety. Thus, conventional internal medicine assessment, focusing on single organ systems or single diseases, is not well suited to the typical older patient, who often has multiple chronic diseases and psychosocial stresses. The interrelation between multiple clinical and psychosocial problems frequently leads to functional impairment. Consequently, clinical disorders in the elderly often manifest as geriatric syndromes, clinical entities characterized by numerous etiologies and complex pathophysiology. Examples of geriatric syndromes included dementia, urinary incontinence, falls, pressure ulcers, and sleep problems. Crashes in the elderly are a similar type of geriatric syndrome, the functional consequence of changing physiology, chronic disease(s) and patterns of behavior.

Influence of Chronic Disease on Driving

Underlying the geriatric syndrome of crashes in the elderly are one or more chronic medical diagnoses. The task is to recognize when illness impairs skills and functions that specifically render a person incompetent for safe driving. Several diseases are highlighted below:

Alterations of Consciousness

Epilepsy, hypoglycemia related to diabetes, cardiogenic syncope, stroke and the excessive daytime sleepiness of sleep apnea can cause sudden changes in level of consciousness that would be disastrous to a driver. Fortunately accident rates due to sudden incapacitation appear to be rare, causing .9-2.1 per 1000 crashes.[8] About half of these accidents are related to a cardiac etiology. Even with a sudden cardiac event, most drivers can avert a major accident. Further discussion on the driver with disorders affecting consciousness appears in Dr. Mernoff's article.

Visual Impairment

Changes in vision are perhaps the most studied, most quantifiable and best predictors of crash risk. With age there is a gradual opacification of the cornea, lens and vitreous. Under identical illumination, the retina of a 60 year-old receives 1/3 the light of a 21 year-old. The ability to notice movement approaching from the periphery is affected most by the decrease in light delivery. Thus, twilight is considered the most difficult time for driving. Increased light scatter on the aging retina causes problems with pattern detection and glare. The ability of the lens to accommodate (focus) declines. In addition to normal changes in the visual system, eye diseases occur frequently with aging. The four most common causes of vision impairment in older adults, according to the Framingham Eye Study,[9] are age-related maculopathy, cataract, glaucoma and diabetic retinopathy: 19% adults between 65-75 have at least one of these conditions, with 50% affected over age 75.

Standard visual tests of acuity and peripheral vision are performed on non-moving or static stimuli. Although studies examining the relationship between static visual acuity and crashes found statistically significant relationships between visual impairment and the number of crashes, the correlation is weak.[10] The data are insufficient to help achieve the practical goal of successfully identifying which older drivers are seriously at risk for a crash. Newer, dynamic measure of visual function, such as the useful field of view (UFOV), are promising predictors of crash risk. Dynamic visual assessments incorporate visual information processing, visual attention,

attention switching, divided attention and visual search. Dynamic visual testing has emerged as an important independent predictor of crash risk in retrospective studies and must now be validated in a prospective manner.

CARDIOVASCULAR DISEASE

Unstable angina, characterized by change in the frequency, duration or ease of provocation of symptoms, should preclude driving. Once angina is stable for 3-4 weeks the elderly patient can drive with an acceptable level of risk.[11] Changes in cardiac medication may require suspension of driving for several weeks to monitor for side effects. Patients with chronic, stable angina should avoid driving conditions which may provoke angina, such as bad weather, hauling a trailer, or sustained driving. General guidelines would include a stop after every 90 minutes of driving with a maximum of six hours per day. Nitroglycerin may induce lightheadedness or syncope and should be used at rest, not while driving.

The approach to activity after myocardial infarction has changed dramatically over the last few decades. Bedrest for many weeks was recommended at one time, while now a treadmill exercise test may be administered within days of infarction. Patients with uncomplicated infarction who are able to perform routine daily activities without symptoms should be able to engage in non-stressful driving by the fourth week. Generally, a waiting period of several days following angioplasty and one month following bypass surgery is advised before resuming driving.[12]

DEMENTIA

Dementia involves impaired memory and other changes in cognition, judgment and personality that interfere with work, family or social activities. Alzheimer's disease is the most common cause of dementia with a prevalence as high as 11% for those over age 65, and 47% for those over age 85.[2] The early diagnosis of Alzheimer's is difficult because some age-related cognitive decline may blend with the early stages of the disease. As a result, many persons with early Alzheimer's dementia are drivers and may not be seen by physicians. A detailed discussion of the driver with dementia appears in Dr. Whelihan's article.

> *... drivers over age 60 and especially over 75 have more right angle crashes and crashes associated with rights of way, intersections and left hand turns.*

MEDICATIONS AND DRIVING

The use of prescription medications, including those suspected of adversely affecting driving, increases with age. The typical older driver is taking medication, with 80% of people over age 65 receiving one or more prescription medications.[13] Medications of particular concern include benzodiazepines, antidepressants, opioids, antihistamines, and oral hypoglycemic agents. The effects of some benzodiazepine on attention and motor coordination can persist even several weeks after discontinuance.[14] A prospective cohort study of drivers aged 65-84 followed for 4 years found a 50% higher rate of crashes in current benzodiazepine users, with crash risk increasing with benziodiazepine dose.[15] Physicians should use benzodiazepines sparingly and avoid prescribing long acting benzodiazepines such as diazepam (Valium®), or flurazepam (Dalmane®) in favor of short half-life alternatives such as tempazepam (Restoril®) or lorazepam (Ativan®). Consider substituting non- benzodiazepine anxiolytics/hypnotics such as buspirone (Buspar®) or zolpidem (Ambien®).

Tricyclic antidepressants also increase crash risk in case control studies of elderly drivers.[16] Nortriptyline (Pamelor®) and desipramine are recommended for elderly patients over amitriptyline (Elavil®) because of the lower incidence of sedation and postural hypotension. Physicians should consider alternate, non-tricylic antidepressants such as the serotonin reuptake inhibitors fluoxetine (Prozac®), sertraline (Zoloft®) or paroxetine (Paxil®), which lack the sedative, anticholinergic and cardiovascular side effects of tricyclics and produced little or no impairment on open-road testing among older subjects.[17] Many antihistamines are sedating and impair coordination. Non-sedating varieties such as loratadine (Clariten®) and fexofenadine(Allegra®) are drugs of choice as they do not cross the blood-brain barrier nor produce performance impairment in simulated driving. Drug-induced hypoglycemia in diabetics is more common with insulin than oral agents. The newer biguanides (Glucophage®) do not induce hypoglycemia when used alone. The effects of opioids on psychomotor function and driving skills in the elderly population have not been well studied. In a Tennessee Medicaid study, the authors did not find significantly increased rates of crash involvement among current users of oral opioids.[16] Less potent opioids such as codeine and propoxyphene (Darvon®) cause less sedation than more potent opioids such as oxycodone (Percocet®). Analgesic efficacy of acetaminophen (Tylenol®) and ibuprofen (Motrin®) are equivalent to low potency opioids without the sedation.

ASSESSMENT OF DRIVER COMPETENCE

Physicians vary widely in their ability, experience and confidence in judging the driving competence and safety of those with medical diagnoses. There is intuitive and general agreement on severely affected patients when driving has become dangerous, but more subtle cases may be difficult to identify with a standard medical history and physical exam. Physicians do not yet have simple, sensitive and specific tools to predict patient fitness to drive. To do a detailed functional assessment, that would uncover significant driving impairment, on all older drivers would be costly and time-consuming, and has not been sufficiently proven with regard to validity and reliability. Certainly, physicians can provide medical input and opinion but ultimately decisions on driving competence are made by state licensing agencies. What is considered acceptable risk and how wide a net do we cast to find impaired drivers? Until these issues are clarified it is prudent to avoid establishment of regulations on elderly drivers on the basis of insufficient knowledge that may not be effective and may impose discriminatory controls.

CORRELATES OF IMPAIRED DRIVING COMPETENCE

Advances in Functional Testing

Direct observation of drivers performing in traffic would seem a good predictor of crash risk. However, driving is an over-learned behavior and a standard road test may not expose inadequacies of driving performance. What is needed is a road test in which driver responses to stressful situations, detours and unexpected obstacles can be assessed. This would be impossible to achieve with a road test, but computer technology allows increasingly realistic simulations of driving. Driving simulator testing has correlated with the on-road driving performance of older adults, and in a retrospective fashion identified drivers at high risk for crashes.[18] What has not been tested is whether driver performance on the simulator predicts occurrence of future accidents. Research on driving simulators is discussed elsewhere in this issue by Susan Martin.

Falls

Falls and motor vehicle crashes share similar risk factors, including cognitive impairment, gait and balance abnormalities, lower extremity disability, neurologic disease, impaired vision and polypharmacy.[19] If prospective studies confirm that falling and car crashes share predictive risk factors, a unified approach to the prevention of these mobility disorders could result. Until more information is available, clinicians should investigate driving competence or refer for further evaluation their elderly patients who fall.

Interventions

Typically, interventions to address elderly, problem drivers are limited and often take place only after some driving infraction or accident occurs. It is crucial to develop interventions which precede accidents. One approach to the prevention of crashes in the older driver is to develop separate measures aimed at the driver, at the vehicle and at the infrastructure (road conditions and traffic management). To improve the driver, physicians must first ask about their older patients' driving habits. It is often useful to ask hospitalized seniors whether they intend to drive after discharge. A study of geriatric inpatients showed that of 37 patients who intended to drive after discharge only five (14%) met the medical requirements of the investigators for safe driving. All the interviewed patients thought themselves safe drivers.[20]

Physicians can also promote driver safety by educating patients about their limitations and suggesting avoidance of night, highway and rush-hour driving. Physicians can take advantage of resources in the community. Local neuropsychology programs perform driver evaluations and the AARP sponsors a 55 Alive/Mature Driving Class. Physicians must ensure that older drivers have their best possible vision and should regularly scrutinize medication lists to minimize driving impairment. Family support can be enlisted to limit or remove access to a vehicle and develop alternate modes of transportation. A serious concern over driver competence can be referred to the Department of Motor Vehicles Medical Review Board. Vehicle enhancements to improve safety include passive safety devices, side impact protection, enhanced power steering, auxiliary mirrors, hand controls, swivel seat, extended steering column, and steering wheel knobs. In the near future, electronic driver support technology such as audible warnings about stop signs, excessive speed or poor road-centering will be available. Infrastructure changes might include designing roadways to reduce intersection speeds, improve road signs, and standardize intersection layouts

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Paroxysmal Disorders and Driving

Stephen T. Mernoff, MD

BACKGROUND

At least 71%, or more, of all car crashes involve "driver error."[1] It is intuitive that disease underlies a significant portion of motor vehicle mishaps, and there is now a substantial body of data to support this assumption. Sjogren et al[2] found that 25% of fatally injured drivers had disease and that in 6% disease was probably the underlying cause of the crash.

Current policies regarding licensing of drivers with medical conditions vary from state to state and country to country. This probably reflects at best a lack of consistent data on which to base policies and at worst social prejudice related to ignorance (epilepsy /driving laws are a prime example). Prospective studies are difficult to do because of ethical/legal considerations and retrospective studies are inherently biased. A better understanding of the role of disease in car crashes could promote the development of rational, evidence-based interventions to reduce the rate of injuries and property loss.

A case report describes an individual with reflex absence epilepsy whose seizures were precipitated by sitting in a car, touching a steering wheel, or thinking about driving. This is an extreme example but illustrates that in some cases the licensing decision is relatively straightforward. However, there are many cases for which the decision is not so easy. The growing body of data can be used to develop evidence-based policies for drivers with medical issues.

This paper discusses some of the data regarding the relationship between some paroxysmal disorders and driving competency. There are limitations to the data, but the data are still useful. Public policy, as it relates to such disorders, is discussed elsewhere in this issue. ("Driving Policy Issues and the Physician")

Abbreviations Used:

AED	anti-epileptic medication
AF:	atrial fibrillation
DMV	Department of Motor Vehicles
DWE	drivers with epilepsy
ICD	implantable cardioverter-defibrillators
LVEF:	left ventricular ejection fraction
MI	myocardial infarction
OSA:	obstructive sleep apnea
PWE:	patients with epilepsy
SVT:	supraventricular tachycardia
VT/VF:	ventricular tachycardia/ventricular fibrillation

SEIZURE DISODERS AND DRIVING

In the US the lifetime risk of having a seizure is about 5% and in most cases there is no recurrence. However, the prevalence of true seizure disorders in the US is about 1%, and many of these individuals drive, often against advice. The results of risk studies of epilepsy and driving are variable and sometimes contradictory, probably because the populations studied vary considerably. Some older studies are obsolete because of advances in the management of epilepsy. However, some patterns do emerge upon review of the literature.

* Risk of seizure recurrence

The risk of a second seizure within 3 years of a single idiopathic seizure is about 30% and the lifetime risk of recurrence is 14-50%.[3] There seems to be a consensus that an individual can drive after a single idiopathic seizure after a seizure-free period (duration controversial) if the neurologic exam, neuroimaging, and EEG are all normal.

For patients with epilepsy (i.e. recurrent seizures), the longer the seizure-free interval, the lower the risk of a future seizure: 85% of PWE who were seizure-free for 3 months were seizure-free for one year; of these 75% remained seizure-free for 3 years.[4] Fisher and Krumholz[5] recommend a 3-month observation/medication adjustment period after a seizure before resuming driving as an appropriate risk:benefit balance in most (but not all) cases.

* Are drivers with seizure disorders at greater risk of driving mishaps?

Studies have shown varying results but often involved different populations and endpoints. Drivers with episodic disorders may have up to twice the risk of crashes and/or moving violations compared to the general population or age-matched controls[6] but other studies have reported epilepsy imparted only mildly increased risk of crashes and moving violations. Still other studies have shown no increase in crash risk, but higher rates of serious injuries, nondriver fatalities, careless driving violations and drug violations among DWE compared to controls.

Hashimoto et al[7] reported that over a 5-year period, 9% of 81 DWE had crashes but none were due to seizures. However, Hasegawa et al[8] surveyed 72 DWE; 25% reported at least one crash due to seizure and 10% of the crashes (mostly minor) were caused by simple partial seizures.

*** Are there identifiable subsets that are at particularly high risk?**

The data support the notion that the overall increase in risk imparted by the presence of epilepsy does not warrant generalized driving restrictions on all DWE. The issue then becomes one of determining which subsets of DWE are at particularly high risk. This would guide appropriate interventions in an effort to limit mishaps.

Risk factors for crashes or moving violations among DWE include young age, male sex, single status, history of multiple seizures, lack of treatment with an AED(s), and history of alcohol abuse.[6] Gastaut et al[9] reported that 82 subjects with well-classified epilepsy reported 109 seizures while driving, and crashes resulted from 55% of the seizures. Presence of aura and no change in mentation (i.e. simple partial seizure) during the seizure reduced the likelihood that a given seizure would cause a crash. The authors proposed granting a license after a one-year seizure-free interval, and granting temporary permits if seizures were without change in mentation and loss of motor control, and if the driver had no history of seizure-related crashes and was compliant with medications.

Better, more controlled data geared toward determining high-risk subsets would be useful in developing driving policy. These studies would be particularly useful: mishap risk vs. seizure type, mishap risk vs. seizure frequency, mishap risk vs. seizure recurrence, and risk of recurrence vs. seizure type.

Public policy relating to driving and seizure disorders is extensively discussed elsewhere in this issue (see "Driving Policy Issues and the Physician").

SYNCOPE

* Cardiac arrhythmias

Patients with SVT or ICDs often continue to drive despite having arrhythmia symptoms or ICD shock while driving or being advised not to drive by their physician.[10] Sometimes physicians do not raise the issue with their patients. The risk of an episode while driving and injury due to an episode while driving is relatively low, but it is not zero. Bansch et al[11] found that among patients given an ICD, 54% had recurrent VT/VF, 10% had a syncopal episode within a year and 19% within 3 years of implantation. Chronic AF, low LVEF, high VT rate and syncope during first VT episode post-ICD placement were risk factors for syncope.

> *In one telephone survey, 23% of drivers admitted to falling asleep at the wheel at least once …*

◆

In the United Kingdom the following guidelines are in place for cardiac patients:[12]

- post-MI, pacemaker insertion, heart surgery or angioplasty: no driving for 1 month (arrhythmia risk)
- symptomatic arrhythmias: no driving, but resume "once these symptoms are controlled" ("controlled" not defined)
- asymptomatic paroxysmal ventricular tachycardia, heart block: no driving until cleared by cardiologist ("clearance" criteria not specified)
- as of May 1995, driving is allowed if an ICD has been in place without discharge for at least 12 months with no prior history of incapacitating discharge

Jung et al[13] calculated, for drivers with ICDs, a 1 in 45,000 annual risk of injury to others. They recommend a tiered approach to driving restrictions for those with ICDs:

- no restriction for those with asymptomatic nonsustained VT
- 6-month restriction for patients without recurrences of ventricular tachyarrhythmias (longer unspecified period for those with well tolerated ventricular tachyarrhythmias)
- total restriction for commercial drivers with ICDs and noncommercial drivers with recurrent unstable ventricular tachyarrhythmias.

HYPOGLYCEMIA EPISODES

Hypoglycemia can cause a range of symptoms, from mild attentional impairments to hemiparesis, severe encephalopathy, seizures and coma. Patients may not be aware of the symptoms when mild. Peripheral neuropathy and treatment with ß-blockers can interfere with recognition by blunting some symptoms. Most diabetics experience hypoglycemia at some point. Ward and Stewart[14] found 40% of a group of diabetic drivers reported hypoglycemic episodes while driving, and 13% reported a mishap while driving due to hypoglycemia.

Older studies indicated an increase in crash risk among diabetics but more recent studies vary from indicating mildly increased risk to decreased risk. Whether diabetes-related static impairments or hypoglycemic episodes are the more important factor has apparently not yet been worked out. I did not come across studies that determined subgroups at greatest risk for hypoglycemia.

Some states require a physician's note stating that a patient's diabetes will not interfere with his/her ability to drive safely. Rhode Island does not have a formal written policy regarding diabetics, but policies are being revised this year. In Massachusetts, the "Seizure and loss of consciousness policy statement" appears to be applicable to hypoglycemic episodes.

SLEEP DISORDERS

Drowsiness while driving is clearly a risk factor for crashes. It is underreported by drivers. The National Commission on Sleep Disorders Research found that drowsiness might be a factor in 42% to 54% of crashes (and in

36% of fatal crashes).[15] In one telephone survey,[16] 23% of drivers admitted to falling asleep at the wheel at least once, 2% crashed while driving drowsy, and 2.8% crashed falling asleep at the wheel. Being male, <30 years old, having limited sleep just before driving, driving long distances, and driving for a job are predictive of drowsy driving. Peak incidence is in late afternoon and early morning.[17]

Obstructive sleep apnea and narcolepsy are specific causes of daytime drowsiness that lead to increased risk of crashes and impaired laboratory driving performance but data is limited. I have not come across literature attempting to identify high-risk subgroups; such data could be helpful to physicians in regards to both treatment and advising patients regarding driving.

OBSTRUCTIVE SLEEP APNEA (OSA)

OSA is usually associated with sleepiness and poor daytime concentration. Studies in driving simulators show reduced attention, increased reaction time, and periods of sleep among persons with OSA. Analyses have shown a two- to three-fold increased crash risk for persons with OSA. A recent case-control study[18] showed a strong association between sleep apnea and crash risk, even when controlling for many potential confounding variables. Uvulopalato-pharyngoplasty has been shown to improve attention and driving performance for at least 3-4 years.[19] Thus, crash risk for drivers with OSA can be added to the list of reasons to fully work up and aggressively treat individuals complaining of drowsiness.

NARCOLEPSY

Symptoms of narcolepsy which increase driving risk include excessive daytime sleepiness, sudden sleep attacks, cataplexy, and sleep paralysis. Cataplexy and sleep paralysis do occur while driving. Compared to controls, narcoleptic drivers are at greater risk of drowsy driving and of falling asleep at the wheel, and of crashes due to these symptoms.[19] Narcoleptic patients, like patients with seizure disorders, sometimes avoid treatment because they fear losing their licenses. Driving risk may be higher for narcoleptic than epileptic patients, probably because of the nonparoxysmal symptoms (i.e., excessive daytime sleepiness).

CONCLUSIONS

To keep crash risk among DWE and other paroxysmal disorders in perspective, in 10,000 crashes, 500 are attributable to alcohol abuse, 100 to males<25 years old, 6 to sudden natural death at the wheel, and 1 to a seizure. From a legislative standpoint, clearly alcohol and drowsiness are much bigger issues than epilepsy and other paroxysmal disorders. However, for many physicians, making driving recommendations for those with medical problems is a daily issue.

Approaching the problem as a risk:benefit ratio analysis is useful. The "risk" assessment is largely a matter of identifying subgroups of patients at greatest risk. Although there is more subgroup data available for seizure disorders, the broad principles are generalizable to other paroxysmal disorders (see AAN guidelines in "Driving Policy and the Physician"). Frequency of episodes, degree of disability of episodes, presence of aura or other warning, number of miles driven per week, types of roads (traffic density, etc.), availability of alternative transportation, and compliance with treatment are just some of the factors involved in making what clearly needs to be a highly individualized recommendation.

Lessons already learned from studying driving in the setting of various medical conditions (particularly epilepsy) are allowing the development of informed, data-driven policies applicable to individuals with static and paroxysmal disease. Physicians in Rhode Island and Massachusetts should be pleased that both states are currently revising their policies with this information in mind. The policies must be flexible enough to allow for consideration of all factors involved. Because of the multifactorial nature of the risk:benefit analysis, care must be taken to avoid rigid formulaic decision-making and to include input from physicians familiar with the complexities of these disorders.

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The Driver with Dementia:
A Role for the Neuropsychologist

William M. Whelihan, PhD, and Margaret A. DiCarlo, PhD

The automobile, for better or for worse, has become a crucial ally in our day-to-day functioning. Even older individuals who have moved beyond the demands of work or shuttling children are adamant about continuing to drive, if only to the local grocery store or senior center. When asked, they will say they want the freedom to come and go as they please, the independence from reliance on relatives or friends.

Driving does enhance autonomy but also, unfortunately, poses safety risks. Manipulating this ever more sophisticated piece of machinery in a fast-paced environment requires quick reactions and the ability to process multiple stimuli simultaneously. Unfortunately, these critical cognitive skills become compromised with aging: reaction time is slowed and it is more difficult to focus on more than one thing at a time.

Elderly drivers are at higher risk than all other age groups for involvement in motor vehicle accidents (MVA) on a per-mile basis,[1-3] consequent to age-related changes in cognitive, sensory, and motor functions[1,3] as well as increased medical complications.[4] When an older person suffers dementia, there are disturbances in a broad range of cognitive functions and a deterioration in previously learned abilities, especially complex tasks. Unfortunately dementia, even in early stages, can also affect insight and judgment so that these cognitive impairments are not recognized and appropriate modifications are not self-instituted. While most investigators and professionals agree that persons with advanced dementia are at increased risk for hazardous driving,[3,5-7] there is no consensus on whether or when individuals with mild or questionable dementia should have restrictions on driving. This latter group presents a major challenge to family members and professionals. Deficits are mild and perhaps inconsistent, and the primary care physician cannot easily differentiate an early dementing process from what might be normal aging.

Abbreviations Used:

DAT	dementia of the Alzheimer's Type
DMV	Division of Motor Vehicles
MMSE	Mini Mental Status Examination
MVA	motor vehicle accident.
UFOV	Useful Field of View

The neuropsychologist, in collaboration with other healthcare professionals, is in a unique position to assist in the diagnosis of early cognitive decline, to determine the severity of neurocognitive and neurobehavioral dysfunction, to track progression of impairment over time, to identify patterns of cognitive strengths and weaknesses that may predict functional impairment, and to assist individuals and families in negotiating the delicate balance between safety and autonomy, particulary in regard to driving.

> *Families often witness less-than-perfect driving behaviors and are torn between allowing an older relative to drive and instituting restrictions ...*

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Despite the well-established value of neuropsychological assessment in the diagnosis of early cognitive decline, the utility of neuropsychological measures in the determination of driving competence has not been researched adequately. Surprisingly few studies have examined the role of neuropsychological functioning as an early indicator of potentially unsafe driving. Findings from the limited studies that have examined the relationship between cognitive factors and driving ability are inconsistent, likely reflecting the varied mental status and neuropsychological instruments utilized, the differing criterion measures of driving competence, and the small sample sizes and heterogeneity of patient populations.

Studies examining the relationship between Mini Mental Status Examination (MMSE) scores and driving ability have been equivocal,[8,9] as might be expected using a single, broad test that has been shown to be insensitive to early-stage cognitive loss.[10] As such, Duchek and colleagues[11] suggested that cognitive screening measures should be used to identify those individuals at risk for driving impairment and in need of more thorough evaluation of the specific skills and processes that influence driving performance.

Investigators who employed specific neuropsychological tests have reported inconsistent results. The majority of these studies either restricted their focus to measures of memory, language, and visuoperception, while neglecting to examine the role of specific executive functions, or they utilized executive measures that may not detect the specific type of dysfunction that affects driving ability.

Executive functions such as planning, problem-solving, response initiation/inhibition, and sustained, selective and divided attention have intuitive appeal as factors related to safe driving. However, they have not been explored adequately to date. One notable exception is the research on visual attention.

Ball and colleagues[12] developed a computer-administered test, Useful Field of View (UFOV), to determine the size of the visual area available in a single glance (without head or eye movements) for a particular task. The UFOV assesses speed of visual processing, central identification and peripheral localization of target stimuli, and divided and selective attention. This measure has shown promise as a correlate of driving ability. For example, using logistic regression models, Sims and associates[13] found that a 40% reduction in useful field of vision was associated with increased crash involvement. In addition, Owsley and colleagues[14-15] reported that statistical models incorporating the UFOV and general mental status are useful in predicting accident frequency with samples of older drivers.

Results of a more recent investigation by Duchek and colleagues[11] revealed that performance on tasks of visual search and selective attention was predictive of on-road driving ability above and beyond dementia severity ratings and several traditional psychometric tests. Specifically, these authors found that error rate in visual search (ability to select a target in a visual array) accounted for 19% of the variance in their entire sample of healthy controls and patients with mild dementia of the Alzheimer's type (DAT), as well as 30% of the variance in their DAT sample. Overall reaction time in visual search and performance on the Boston Naming Test also contributed significantly to the prediction of driving performance. Moreover, these authors demonstrated the value of error type (false positive or false alarm) on a test of visual monitoring (ability to detect change in visual display) in predicting on-road driving performance in individuals with mild DAT.

It is interesting to note that while UFOV performance was significantly correlated with on-road driving performance in the study conducted by Duchek and colleagues,[11] it was far too difficult for the majority of the individuals in their DAT sample to complete. The authors proposed that a simpler version of the UFOV would be necessary to adequately assess its predictive utility. Thus, at present, the UFOV appears to be sensitive enough to discriminate between patients with dementia and healthy controls. However, it may lack the specificity to determine what particular aspect of complex visual attention is most useful in predicting driving performance in individuals with early-stage dementia.

While most investigators and professionals agree that persons with advanced dementia are at increased risk for hazardous driving, there is no consensus on whether or when individuals with mild or questionable dementia should have restrictions on driving.

Although many of the psychometric measures utilized by Duchek and colleagues[11] were not useful in predicting on-road driving performance, these authors focused on measures of memory, perceptual motor performance, and language, to the exclusion of a variety of executive measures. In fact, these authors included only two putative measures of executive functioning (Trail Making test, Part A and Mental Control subtests from the Wechsler Memory Scale) that merely measure speed of processing, simple sequencing, and simple/sustained attention. Such skills may be necessary for safe driving, but they are probably not sufficient.

Another critical issue regarding identifying older drivers at risk is establishing the validity of family members' appraisal of driving competence. Families often witness less-than-perfect driving behaviors and are torn between allowing an older relative to drive and instituting restrictions to insure the safety of their loved one and others. It is often difficult for family members to be objective, and it would not be unusual for them to avoid a confrontation, particularly one that demands change. The issue is multidimensional. On the one hand, family members' ratings of driving competence may be more ecologically valid, given the multiple opportunities for observation under typical, rather than simulated conditions afforded by one-time road test evaluations or driving simulators. On the other hand, the potential psychosocial consequences of restricting a loved one's independence may inhibit accurate appraisal or a family's search for a solution.

As such, healthcare professionals, in collaboration with one another, are in a position to assist families as they negotiate the autonomy-safety issues with an impaired older relative. For example, physicians may choose to refer elderly individuals who demonstrate early signs of cognitive decline (through clinical evaluation, general screening measures, and subjective/collateral reports) for a thorough evaluation of neuropsychological functioning. In turn, neuropsycho-logists can provide objective data, individualized recommendations, and supportive intervention to families. This process would likely alleviate the guilt and burden often experi-

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enced by families, and it would assist them in making an informed decision. Moreover, this process would provide healthcare professionals with a firmer basis to make decisions about such a complex and somewhat ambiguous issue.

To further elucidate the role of neuropsychological assessment in the determination of driving competence in older persons and to resolve the inconsistencies in the research to date, a novel study at Roger Williams Hospital[16] is in progress, funded by the National Academy of Neuropsychology. Based on data from a recent pilot project,[17] in combination with investigative considerations highlighted thus far, the project will include the following: a) age-matched samples of normal controls and older persons with questionable or mild dementia (separated by presumed etiology); b) comprehensive neuropsychological assessment, with an emphasis on executive measures and computer-assisted tests of complex visual attention; c) utilization of multiple predictors of driving ability (cognitive measures and family/loved onesí ratings); d) utilization of multiple criterion measures of driving ability (standardized on-road evaluations, self-report measures of driving history, retrospective and prospective records of moving violations and collisions from the DMV); e) examination of level and pattern of performance on neuropsychological measures; f) multiple evaluations over time to track cognitive and functional decline (in cases of continued, restricted driving); and g) examination of the role of supportive, educative, and behavioral interventions with families and individuals struggling to affect a balance between issues of autonomy and safety. Recommendations to family members post-testing will be individualized based upon the pattern of neuropsychological strengths and deficits. Moreover, follow-up contacts with family members and participants will provide data on the implementation of such interventions.

This project seeks to identify an empirical formula, from various sources of objective data, that predicts driving competence in older drivers with questionable or mild cognitive impairment. Eventually, a battery of cognitive measures will be established that is cost-effective and easy to administer, and that discriminates elderly drivers who may require restricted or terminated privileges from those individuals who remain competent to drive safely. Moreover, a collection of various strategies, recommendations, and interventions will be established to assist individuals and families as they deal with changes in cognitive, behavioral, and affective functioning.

The good news in this area is that many older individuals understand the risks and limitations that come with aging, and they make decisions to restrict their driving accordingly. Those who note changes in their driving efficiency may drive only in familiar areas, in fair weather, and during daylight hours. The more worrisome group that requires attention, and perhaps legislation, are those elderly who are beginning to experience early-stage dementia, who might consequently lack insight, and who insist on the status quo for driving. These individuals do not self-restrict and present major problems to family members and to society at large. The neuropsychologist, as a member of the healthcare team, can play an important role in both the assessment of these elderly individuals and in helping family members cope effectively with the impaired older driver.

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Functional Assessment of Driving Competence

Susan A. Martin, OTR/L, BCN

Abbreviations Used:

DPT	Driver Performance Test
MVPT	Motor Free Visual Perceptual Test
UFOV	useful field of view

We take the privilege of driving for granted. In fact, we often consider it a right, rather than a privilege. Driving enables us to pursue vocational as well as leisure interests. But even more importantly, driving empowers us with freedom and independence which enhances our self-esteem. Driving encompasses a wide variety of skills that include vision, cognitive and perceptual abilities, and motor skills. It is an extremely complex task that is rarely, if ever, routine.

Despite the importance of driving, little thought is given to its complexity and the skills required for safe performance. When people suffer an injury, such as a stroke or other cerebral injury, or begin to age, these thoughts appear and one's competence is called into question. Physicians are looked to when concerns arise over an individual's ability to continue to drive. However, the evaluation of these skills does not typically fall into most physicians' area of expertise. Therefore physicians must rely on the clinical expertise of others, usually occupational therapists, who perform driving evaluations. Driving evaluations typically consist of two parts: a pre-driving clinical assessment and an on-the-road evaluation. Occupational therapists may do one or both parts. The on-the-road evaluation may also be performed by a driving instructor from a driving school.

The pre-driving assessment examines the component skills necessary for driving. For the assessment of cognitive and perceptual components, paper and pencil tasks (both standardized and non-standardized) are utilized. Some of the tests that may be included are Trails A and B, sub-tests of the WAIS-R, Motor Free Visual Perceptual Test (MVPT), Hooper Visuospatial test, letter cancellation tests, the Rey-Osterrieth Complex Figure Design Copy, and knowledge of traffic signs.

There has been concern about the usefulness of this type of psychometric testing as a means to determine driving competence. Some critics identify the training environment as being too small compared to that required during driving where head turning and use of peripheral vision is imperative.[3] Others feel that these tests do not adequately emphasize multisensory task performance; that is, coordination of the visual, auditory, and tactile systems is not required as it is in driving.[3] Katz et al. point out that driving requires one to anticipate precarious and potentially harmful situations and that these tests are incapable of eliciting these reactions.[4] While these criticisms have some validity, the most significant problem with psychometric assessments is the absence of standardization of the tests and procedures used between evaluators or programs; therefore there is no reliability between evaluators.[5]

This is not to say that these tests should be abandoned. Psychometric testing serves an important function when performed by a trained clinician and when part of a comprehensive evaluation. This assessment should be the first portion of the evaluation process. Psychometric tests can quantify deficits in cognition and perception. Patently unsafe drivers need not progress to an on-the-road evaluation, where the risks of harm are obvious.[1] Those impaired individuals are also spared the expense and time of a road test.[5]

Psychometric testing has a diagnostic capacity.[5] The tests will yield specific reasons for the breakdown of driving performance, which may then be used to develop a training program to remediate the problem.[5] A study by Galski, Bruno, and Ehle demonstrated that this training was indeed effective in improving deficient perceptual skills, which, in turn, led to improved driving skills.[5]

Two other important, though not clinical, considerations for these psychometric tests, merit attention. Psychometric tests require far less equipment and space and are also much more cost-effective than driving simulators or on-the-road driving evaluations.

The Driver Performance Test (DPT) is used in some pre-driving evaluation programs. The DPT uses a series of videotaped scenes of potentially dangerous driving situations. Clients are asked to view a scene and then respond to a multiple-choice question, selecting from four choices on the best way to respond to that situation. Reactions must be quick because the viewer has only seconds before the next scene.[12]

To reiterate, the greatest drawback to these tests is the lack of standardization. Knowing this, it behooves physicians and the clinicians involved in these programs to establish an open dialogue, so that the physician understands and appreciates what the tests are measuring, and the interpretation of the results.

Visual testing has traditionally been more of a screening, which examined acuity and peripheral vision. More current studies reveal that these measures are not adequate in identifying drivers at risk for car crashes.[6] The literature cites two other variables, selective visual attention and divided attention, as being strong predictors for car crashes.[6]

Ball, Owsley, and colleagues, who have studied vision and its relationship to driving extensively, have identified a visual area which they call the "useful field of view (UFOV)," or that area of the visual field over which one can use rapidly-presented visual information.[6,7] The "useful field of view" relies on both selective and divided attention.[6]

The UFOV is measured with the Visual Attention Analyzer, Model 2000, a computer-based instrument which uses three sub-tests to measure speed of visual processing, ability to divide attention, and selective attention abilities. The results are expressed in terms of the percentage reduction (0-90%).[6] A study by Ball and colleagues showed that older drivers with a 40% or more reduction in their UFOV were 2.2 times more likely to incur a car crash.[7] The UFOV has been shown to have high sensitivity and high specificity in predicting which older drivers had a history of crash problems.[6] In fact, Cox and Taylor stated that the UFOV shows "superior performance" in prediction of risk for car crashes.[8]

Another asset to the Visual Attention Analyzer is that it has a training model. Training programs were found to have promising results. Some older adults had a partial reversal of their deficits. This resultant improvement was still present one year after the completion of the training program.[6] Another tool that shows promise for improving visual deficits is the Dynavision.[13] This apparatus was designed to evaluate and train visuomotor speed and coordination, visual scanning, and visual attention. Preliminary studies have shown improvement in these skills in persons who have sustained traumatic brain injuries or stroke.[13] The Dynavision has also been shown to have high test-retest reliability.[13] More research is needed to establish the strength of the correlation between these training programs and the ability to be a safe, successful driver.

The evaluation of motor skills is a far easier task. Standard assessments of joint range of motion, muscle strength, sensation, and coordination are administered. The requirements are clearer with regard to these physical/motor components than they are with the cognitive and perceptual components; therefore, less discussion in the literature surrounds them. For those individuals with limited motor skills, adaptive equipment will allow most of them to drive. Recommendations can be made immediately. On-the-road training in the use of the equipment is essential.

A second component to some pre-driving clinical evaluations is the use of a driving simulator. Early driving simulators were video game-like scenarios. At best, they had modest realism.[9] The modern versions of driving simulators use the same technology as airplane simulators to create realistic scenarios. For example, the Iowa Driving Simulator can display multiple types of roadways, traffic signs, traffic conditions, and vehicles. The examiner has great discretion as to what obstacles will be presented. The individual is seated in an actual vehicle cab complete with visual display, controls, and a motion control base. Normal motions of driving felt with acceleration and deceleration are possible. Realistic audio effects are also present to create as real an environment as possible.[9] Rizzo, in a study on the use of simulators with the Alzheimer's population, concluded that simulators have great potential as a tool which can standardize driving assessments.[9]

Driving evaluations typically consist of two parts: a pre-driving clinical assessment and an on-the-road evaluation.

Driving simulators are, obviously, safer than an on-the-road evaluation. However, that safety and security are also their drawback. Simulators are two-dimensional and lack the interactive component of driving.[5] The safe environment may alter an individual's behavior (i.e. is this person a risk-taker?) and subsequent driving performance (will the client take more risks because this is perceived as a "game" or is the individual more conservative in a controlled environment?). The need for timely reactions may be affected as the consequences for failing to react are nullified in a simulated environment.[1,10,11] More research is clearly warranted into the usefulness of simulators as a reliable predictor of driving performance.[11] Simulators are costly and, at this time, are not the norm in pre-driving assessments.

The on-the-road assessment would be the final portion of the evaluation process. Unfortunately, this is another area without uniformity. Some instructors use a closed course as part of their assessment. But in order to make a more accurate assessment, driving in traffic is imperative.[4] On-the-road evaluations take one to two hours. Individuals are challenged with a variety of tasks, such as right and left turns, changing lanes, varying speeds, and varying degrees of traffic. Complex situations are not always encountered, however, and therefore predicting a person's ability to react to them cannot be objectively measured.[5] Given the potential complexity of this task, some authors have actually advocated for two on-the-road evaluators to ensure the most thorough evaluation and to ensure that nothing is missed.[1]

Driving conditions vary with time of day, day of week, weather conditions, other drivers, emergency situations (car accidents, emergency vehicles in area, etc.), and our physical and mental condition for that day. Being fatigued, ill, or anxious all influence our actions.[4] It is impossible to test an individual in such a wide variety of situations. Given that, the importance of the pre-driving evaluation increases, as it can predict behaviors and performance with a wide range of tasks examining a variety of components. The combination of both evaluations will present the most complete picture.

It is the clinician's responsibility to

CURRENTLY, there are three driving evaluation programs in Rhode Island. Rhode Island Hospital offers a pre-driving clinical evaluation and has strong working relationships with several local driving schools for the on-the-road evaluation. For more information or to make a referral, contact Susan Martin, OTR/L or Lynne Benavides, OTR/L. Kent Hospital recently developed a driving program consisting of both the clinical assessment and the on-the-road assessment. To make a referral there, contact the Occupational Therapy Department (phone: 736-4298). The Driver Assessment Program at South County Hospital also provides clinical and on-the-road assessments. To make a referral there, contact Linda Curry, Occupational Therapy Clinical Coordinator, (phone: 789-2044).

provide a thorough, objective evaluation which illustrates an individual's mental and physical fitness to drive. From this, a physician can make a recommendation on the patient's fitness for driving. These evaluations will become more valuable if and when the laws require physician to report at-risk drivers. A clinician cannot guarantee, however, that these individuals will exercise the best judgment and use of these various skills while driving, despite a good performance on the evaluation.[5] It is recommended that standardized assessments be developed. There needs to be more consistency between evaluations and evaluators so that the essential skills necessary for this activity are accurately and thoroughly assessed. This uniformity in the evaluation procedures will also lend itself to further research in order to identify what other essential skills and/or behaviors are required for safe driving.[1]

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The Prevention and Treatment of Pressure Ulcers in RI Nursing Homes

David R. Gifford, MD, MPH

Pressure ulcers are relatively common in the nursing home setting occurring in 1% to 30% of residents depending on the type of patients and facility.[1,2] On average, 13% of nursing home residents will develop a pressure ulcer over a two-year period.[1] Pressure ulcers are associated with significant pain, infection, and increased hospitalization and length of stays. Approximately 40% of individuals with pressure ulcers die within one year.[2]

National evidence-based guidelines developed by the Agency for Health Care Policy and Research (AHCPR),[3,4] and the American Medical Directors Association,[5] along with findings reported in the medical literature,[2,6,7] support the use of approaches that can improve the prevention and treatment of pressure ulcers. The guidelines contain recommendations about risk assessment, skin care, pressure relieving methods, and treatment approaches. For example, the AHCPR guidelines recommend that all admissions should be assessed for risk factors related to the development of pressure ulcers and that treatments for pressure ulcers should avoid agents toxic to wound healing (e.g. Betadine, Dakin's, and alcohol).[3,4]

Rhode Island Quality Partners (RIQP) is one of three Peer Review Organizations (PROs) participating in the Northeast Pressure Ulcer Consortium project. The goal is to improve the prevention and treatment of pressure ulcers in nursing homes. The PROs in each state worked with each nursing home to develop a CQI team that could implement AHCPR guideline recommendations about the prevention and treatment of pressure ulcers. RIQP invited all nursing homes in Rhode Island with a minimum of 80 beds and computerized MDS data since January 1998 to participate in this project. Of the 23 facilities that expressed an interest, nine homes were selected to provide a representation of geographic distribution (urban vs rural), ownership status (for-profit vs not-for-profit) and prevalence of pressure ulcers. These facilities worked with RIQP staff to collect data on their risk assessment practices and treatment approaches during a three-month period in the fall of 1998.

Each facility abstracted information from their medical records for the past 20 admissions about their assessment of risk factors for pressure ulcers. Key assessment elements included: assessment of bed and chair mobility, bowel and bladder continence, feeding ability, history of pressure ulcer, and evidence of malnutrition (weight loss and current weight). Of the 174 admissions evaluated, staff assessed most admissions for incontinence (90%) and bed mobility (72%). However, residents were less often assessed for chair mobility (53%), a history of pressure ulcers (25%), or weight change (6%).

Abbreviations Used:

AHCPR	Agency for Health Care Policy and Research
CQI	Continuous Quality Improvement
MDS	Minimum Data Set
PRO	Peer Review Organization
RIQP	Rhode Island Quality Partners

Using MDS data, we identified up to five residents who were at high-risk for developing pressure ulcers but were reported to be free of pressure ulcers. These residents then had a thorough skin inspection. Unexpectedly, many of the facilities discovered at least one pressure ulcer among these high-risk residents. Overall, 34% of all the residents at high risk had a pressure ulcer discovered during the skin inspections. Nearly all the pressure ulcers were stage I (94%), of which more than 90% occurred on the heals. This data suggests that physicians should be vigilant in conducting thorough skin inspections on their high-risk patients, paying particular attention to the heels of their immobile patients.

Each facility also collected information about the treatments used for the last 10 residents with a pressure ulcer (note: not all facilities had 10 residents with a pressure ulcer). Overall, most elements of care were consistent with guideline recommendations. For example, only 14% of residents with a pressure ulcer had a physician order for an inappropriate skin cleanser or toxic agent such as Betadine. Repositioning, as recommended by the guidelines, was done infrequently. While 62% of residents with a pressure ulcer had a plan to reposition them every two hours while in bed, none had a plan for repositioning while in a chair. Similar results were also found for long-term residents at high risk for pressure ulcers. Thus, physicians should review the care plan for residents with pressure ulcers and those at high risk to ensure that residents are being repositioned frequently while in bed and in a chair and that toxic agents are not being used.

Based upon these results, most of the CQI teams in each facility implemented changes to improve their assessments for and treatments of pressure ulcers. We are currently evaluating the impact of these changes. The results also point to several areas that physicians should focus on when seeing their nursing home patients in order to reduce the development of pressure ulcers and improve wound healing.

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Images in Medicine

Aortic Rupture

Peter T. Evangelista, MD, and John J. Cronan, MD

A 20 year-old man presented after sustaining blunt chest trauma in a high-speed motor vehicle accident. Initial chest radiograph demonstrated a widened mediastinum suggestive of mediastinal injury and a left pulmonary contusion. A contrast CT of the chest demonstrated a pseudoaneurysm involving the proximal descending aortic arch (Figure 1, arrow), fluid in the mediastinum, and the left pulmonary contusion. An aortic arch angiogram further demonstrated a traumatic transection of the isthmic portion of the thoracic aorta with the presence of a pseudoaneurysm (Figure 2, arrow).

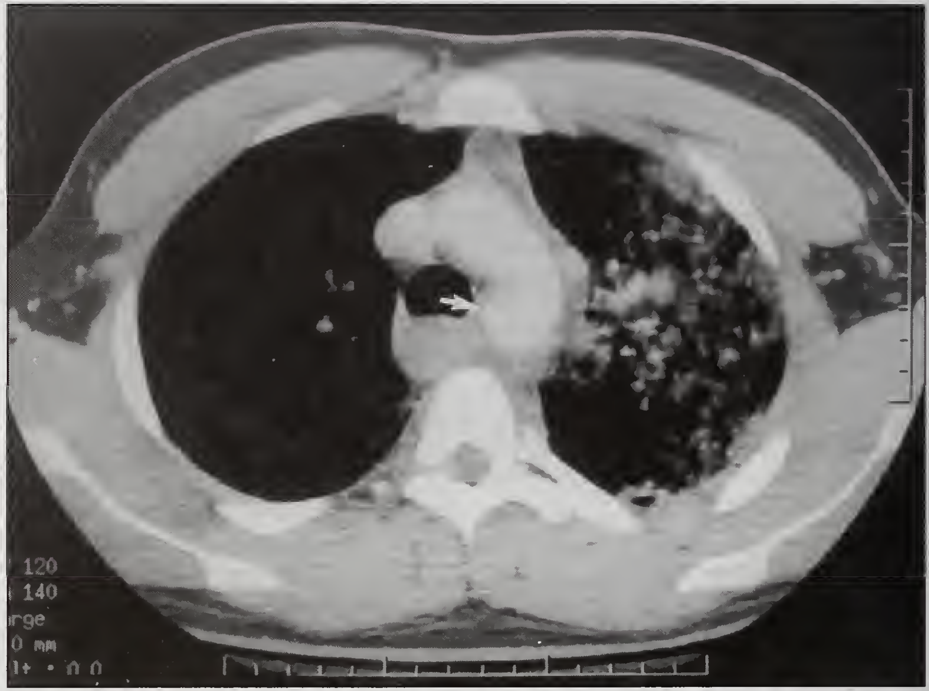

Figure 1.

Reference

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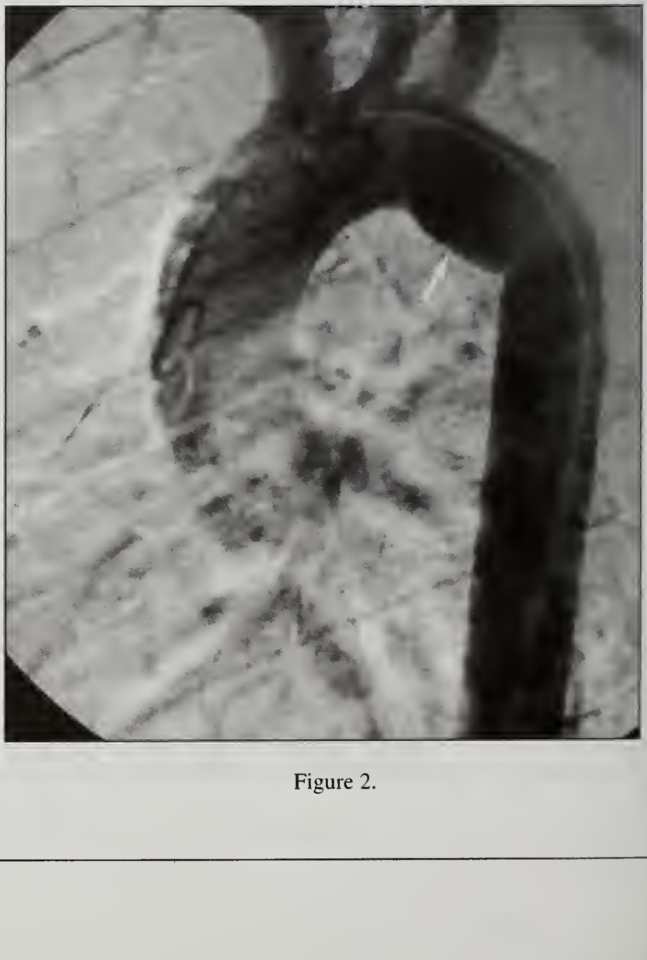

Figure 2.

Health by Numbers

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Jay S. Buechner, PhD

Motor Vehicle Crashes Among the Elderly, 1994-1998

Jay S. Buechner, PhD

National data show that there is an increasing number of licensed drivers ages 70 and older. Between 1987 and 1997, the most recent year for which data are available, the number of older drivers rose 45%.[1] There has been a corresponding increase in the number of older drivers involved in fatal crashes. Nationally, over the ten years ending in 1998, the number of drivers age 70 or older involved in such crashes increased 33%, compared to a decrease of 12% among those under age 70.[1] In this analysis, recent Rhode Island data on motor vehicle deaths and hospitalizations among the elderly are presented.

Methods

Deaths of state residents with an underlying cause of death listed as motor vehicle traffic crashes were tabulated from the Rhode Island death certificate file for the period 1987-1998. (Data for 1996-1998 do not include all out-of-state deaths of Rhode Island residents and are therefore provisional data.) Inpatient stays in the state's acute care hospitals due to motor vehicle injuries were similarly tabulated from the statewide hospital discharge data system for the period October 1, 1993-September 30, 1998, corresponding to hospital fiscal years 1994 through 1998. For both deaths and hospitalizations, persons injured in motor vehicle crashes who were not occupants of passenger cars and trucks (e.g., pedestrians, motorcyclists, and persons riding other vehicles) were excluded from the analysis. Rates were computed using population estimates for Rhode Island obtained from the Bureau of the Census.

Results

During the five-year period 1994-1998, there were 279 deaths of Rhode Island residents due to motor vehicle crashes in which they were drivers or passengers. Of these, 17 (6.1%) were persons ages 70 and older. Similarly, during the five-year period consisting of hospital fiscal years 1994 - 1998, 3,121 persons were hospitalized in Rhode Island due to injuries suffered in motor vehicle crashes in which they were drivers or passengers. Of these, 458 (14.7%) were persons ages 70 and older.

The death rate for motor vehicle occupants was highest for persons ages 15-69, followed by those ages 70 and older and those under age 15. (Figure 1) The rate of hospitalization for injuries caused by motor vehicles was highest for persons ages 70 and older, for whom the rate was 8% higher than for persons ages 15-69 and approximately five times the rate for children under age 15.

When analyzed by more detailed age groups, hospitalization rates for motor vehicle crashes exhibit a bimodal pattern; the highest rates are for person ages 15-19 and ages 20-24, but there is also a clear secondary peak at ages 80-84. (Figure 2) The age-distribution patterns for all occupants and for drivers only are similar, except that drivers form a somewhat smaller percentage of the injured among the age groups where hospitalization rates are highest.

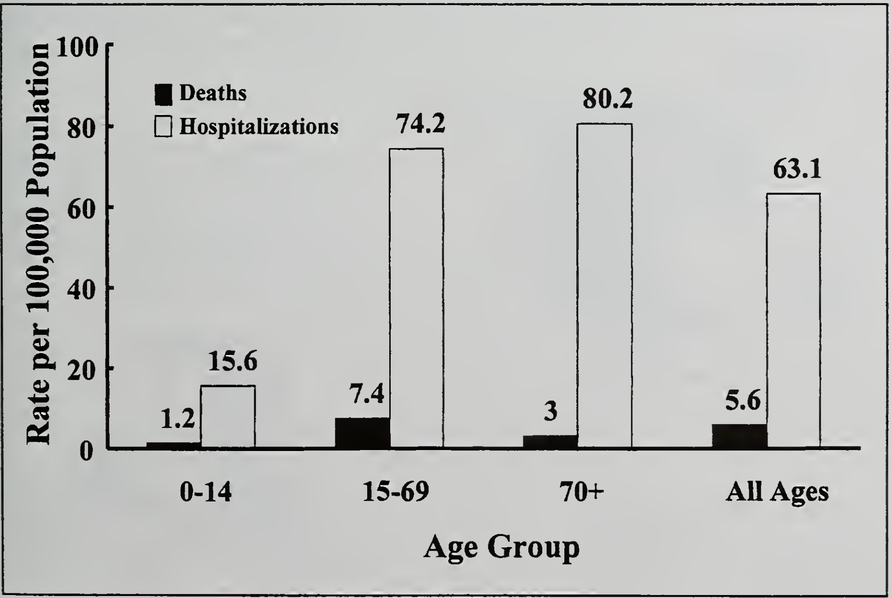

Figure 1. Death and Hospitalization Rates for Occupants in Motor Vehicle Crashes, by Age Group, Rhode Island, 1994 - 1998.

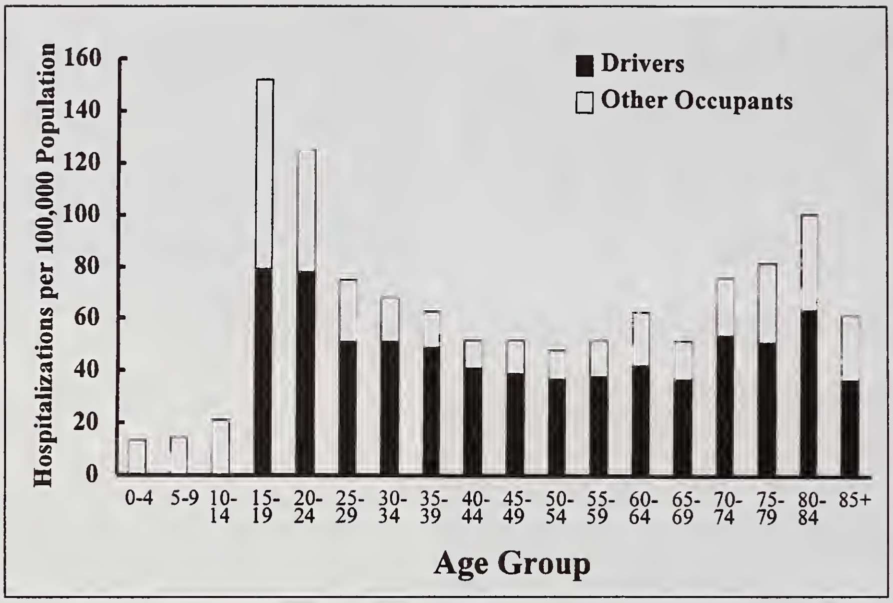

Figure 2. Hospitalization Rate for Occupants in Motor Vehicle Crashes, by Age Group and Position in Vehicle, Rhode Island, 1994 - 1998.

There are differences between younger and older drivers in the types of injurious motor vehicle crashes they are involved in. (Figure 3) A larger proportion of older drivers were injured in collisions with other motor vehicles and a smaller proportion injured in collisions with fixed objects either on or off the roadway (bridge abutments, guardrails, utility poles, etc.). Older drivers are also relatively less likely to be injured in non-collision crashes involving loss of control, such as vehicle roll-overs.

Discussion

The increasing number of older drivers and their high rates of injury from motor vehicle crashes present a complex problem for public health and traffic safety. Traditional programs targeted at safety belt use and alcohol involvement will not yield further substantial changes among this group, as the elderly are already the most likely of all age groups to use safety belts and the least likely to drive after drinking.[2] There are other factors, more specific to this age group, that affect their driving safety, including increased reaction times, cognitive impairments, loss of physical strength, and increased vulnerability to trauma.

Over ten years ago, in September 1989, the Rhode Island Department of Elderly Affairs, with funding from the Department of Transportation, hosted a statewide conference on elderly driving entitled, "License to Drive - Is It Forever?" Subsequently, the Task Force on Older Drivers that was established at the conference presented a proposal including twenty-seven specific recommendations.[3] These recommendations covered the areas of (1) signing and road design, (2) education and training, (3) testing, (4) insurance and legal issues, and (5) alternative transportation. The breadth of this proposal reflected the complexity of the issues surrounding elderly drivers, and most of the recommendations presented then remain relevant a decade later.

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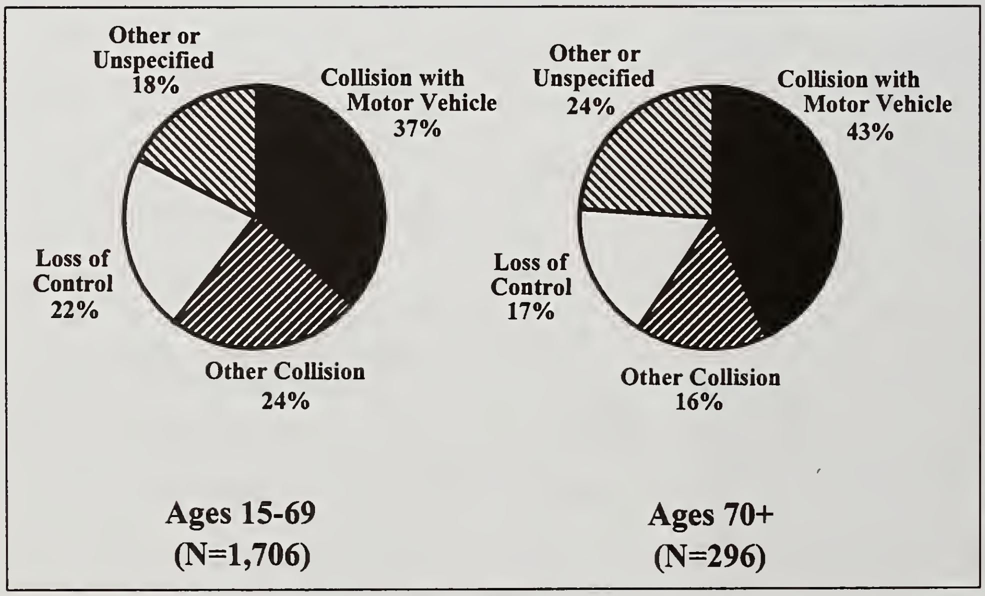

Figure 3. Distribution by Type of Motor Vehicle Crash for Hospitalizations of Drivers in Motor Vehicle Crashes, Rhode Island, 1994 - 1998.

PUBLIC HEALTH BRIEFING

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by John P. Fulton, PhD

Breast Cancer Control Report Card: Rhode Island, 1999

Donald K. Perry, MPA, Sharon Marable, MD, MPH, and Dorothy Darcy, AS, CTR

The Rhode Island Women's Cancer Screening Program (WCSP), funded through the Centers xxfor Disease Control and Prevention (CDC), provides no cost mammograms, Pap tests, and other services in accordance with accepted standards of care. A Rhode Island woman is eligible for the program if she has no health insurance and her family income is within 250% of poverty level. To evaluate the effectiveness of the program and other statewide cancer control activities, the WCSP and the Cancer Information System (Hospital Association of Rhode Island) have constructed the following breast cancer control report card. The report utilizes current state and national statistics,[1,2,3,4] including data from the Behavioral Risk Factor Surveillance System.

The data indicate that the number of women dying from breast cancer in Rhode Island is decreasing overall. Despite the decrease, the breast cancer mortality rate is still higher for Rhode Island Black women than for Blacks nationally. The mortality rate is also higher and is declining more slowly for Black women compared to White women within the State. Consistent with higher mammogram usage rates, especially among older Black women in Rhode Island, recent improvements in the diagnosis of breast cancer in earlier stages should begin to accelerate the reduction in mortality rates and narrow the gap between the races.

Revised in 1997, American Cancer Society (ACS) breast cancer screening guidelines include an annual mammogram, annual clinical breast exam, and monthly breast self-exam for all asymptomatic women over age 40. The tables illustrate that mammogram screening rates in Rhode Island are well above national medians and have increased since 1990, particularly among younger women (age 40-49). However, recent data suggest that statewide rates may be leveling off among younger White women or even declining among younger Hispanic and, especially, Black women. For older women (over age 50), there has been a substantial increase in screening rates among Whites and Blacks, but a slight decline among Hispanics. Notably, mammography usage also remains much lower among women eligible for WCSP services compared to ineligible women, despite recent improvements.

The data on older women may reflect the positive impact of WCSP and other statewide screening initiatives as well as Medicare's resumption of reimbursement for annual mammography in 1998. However, there is room for improvement in screening rates across all populations. Trends among Hispanics, younger women, and women eligible for WCSP services are particularly disturbing. In light of the ACS guidelines, there needs to be an enhanced, coordinated effort to screen women from these groups through outreach, recruitment, and the further reduction of barriers to mammography. Furthermore, health providers should focus more attention on tracking all women and following up with reminders to assure that annual rescreening occurs. Of paramount importance, an abnormal screening result should lead to adequate and timely diagnostic testing.

Abbreviations Used:

ACS	American Cancer Society
CDC	Centers for Disease Control and Prevention
WCSP	Women's Cancer Screening Program

To reduce the burden of breast cancer even further, Rhode Island health care providers must continue to combine routine screening with appropriate and timely follow-up and treatment to all women diagnosed with breast cancer. Since 1995, participating providers have diagnosed breast cancer in 28 women screened through the WCSP. Although the CDC program does not reimburse providers for cancer treatment, Rhode Island physicians and health care institutions provided necessary treatment to all of these women who sought such care. The providers utilized a financial needs assessment process, which resulted in reduced fees or no cost services. The WCSP supports pending national legislation to extend state Medicaid eligibility for cancer treatment to all women enrolled in the CDC program who are diagnosed with breast cancer.

REFERENCES

1. Ries LAG, Kosary CL, Hankey BF, et al. (Eds). *SEER Cancer Statistics Review, 1973-1996.* Bethesda, MD: National Cancer Institute, 1999.
2. U.S. Public Health Service. *Healthy People 2000.* Washington, DC: U.S. DHHS, 1991.

Table 1: Burden of Breast Cancer Mortality by Race
1991-94 baseline vs. 1995-97 latest (1992-96 for latest US Data)

Female Breast Cancer Mortality Rate (Cases or Deaths per 100,000 Population, Age-Adjusted to the 1970 US Pop)	Base Rt RI	Latest Rt RI	Latest Rt US	Progress RI
All Women	29.6	25.3	25.4	++
White Women	29.7	25.0	25.1	++
Black Women	36.4	34.4	31.3	+

Table 2a: Thoroughness of Breast Cancer Screening by Age
1990 baseline vs. 1996 latest

Mammography Usage Rate (% of women getting mammogram)	Base % RI	Latest % RI	Latest % US Median	Progress RI
Women ages 40-49 getting biennial mammogram	66.5	73.0	65.4	+
Women ages 50+ getting annual mammogram	60.9	62.3	57.0	=

Table 2b: Thoroughness of Breast Cancer Screening by Age and Race/Ethnicity
1994-96 baseline vs. 1996-98 latest

Mammography Usage Rate (% of women getting mammogram)	Base % RI	Latest % RI	Progress RI
Women ages 40-49 getting biennial mammogram among:			
White (Non-Hispanic) Women	73.6	75.1	=
Black (Non-Hispanic) Women	82.7*	64.9*	– –
Hispanic Women	74.1*	67.2*	–
Women ages 50+ getting annual mammogram among:			
White (Non-Hispanic) Women	56.1	64.8	++
Black (Non-Hispanic) Women	65.6*	78.1*	++
Hispanic Women	56.2*	53.3*	–

* Caution: based on small numbers.

Table 2c: Thoroughness of Breast Cancer Screening by Age and RI WCSP Eligibility
1994-96 baseline vs. 1996-98 latest

Mammography Usage Rate (% of women getting mammogram)	Base % RI	Latest % RI	Progress RI
Women eligible for WCSP services getting annual mammogram	30.1*	37.0	++
Women ineligible for WCSP services getting annual mammogram	58.9	66.9	++

† Eligibility: age 50 and over, no health insurance, and family income within 250% of poverty.
* Caution: based on small numbers.

Table 3: Effects of Screening by Race
1987-91 baseline vs. 1992-96 latest

Early Detection Rate (% of female breast cancers diagnosed "in situ" or "localized')	Base % RI	Latest % RI	Progress RI
White Women	64	66	=
Black Women	48	52	+

3. American Cancer Society. *Cancer Risk Report. Prevention and Control 1998.* Atlanta, GA: American Cancer Society, 1998.
4. Chen VW, Wu XC, Andrews PA (eds). *Cancer in North America, 1990-1994. Volumes One and Two: Incidence and Mortality.* Sacramento, CA: North American Association of Central Cancer Registries, 1998.

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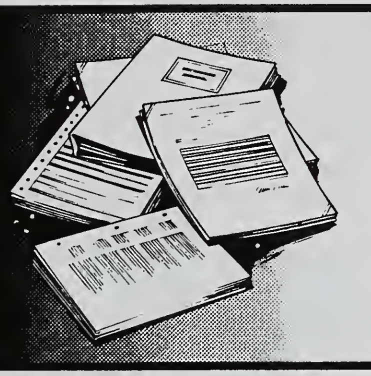

CLINICAL TRIALS DIRECTORY

Medicine & Health/Rhode Island is pleased to publish this Directory. If you would like to list a trial, please contact: Joan Retsinas, Managing Editor.
phone: (401) 272-0422
fax: (401) 272-4946
e-mail: JRetsinas@aol.com.

Topmat Neuropathic Pain Study: A Double Blind, Placebo-Controlled, Parallel Group, Dose-Response Study to Evaluate the Efficacy and Safety of Topiramate Versus Placebo in the Relief of Pain in Diabetic Peripheral Polyneuropathy

Sponsor: R. W. Johnson Pharmaceutical Research Institute

Purpose : Relief of Pain in Diabetic Peripheral Polyneuropathy. The purpose of this study is to evaluate the efficacy and safety of three doses of topiramate versus placebo in the treatment of pain associated with diabetic peripheral polyneuropathy. This study will also evaluate the benefits to health-related quality of life associated with the use of topiramate relative to placebo for the treatment of pain associated with diabetic peripheral polyneuropathy

Principal Investigators: Dr. Norman Gordon, Principal Investigator; Dr. Peter Bellafiore, Sub Investigator; Dr. Vlad Zayas, Sub Investigator

Patients Recruited: Patients who meet the following criteria may be eligible to participate: Pain associated with established (six months) diabetic peripheral polyneuropathy; Adequately controlled diabetes. Patients must be between 18 and 75 years of age

Intervention: The main benefit is the possibility that the patient's health-related quality of life may improve.

Duration: The study will last to up one year with possible extensions.

Phase: IIIb

Sites: CNS Research, Inc., 100 Butler Drive, Providence, RI 02906; Dr. Peter Bellafiore, 70 Kenyon Avenue , Suite 21 Wakefield, RI 02879.

Contact: Jeanne Monast, RN, CRC 1-800-847-4332

e-mail:pepple@cns-research.com

BRAVO Study: Blockade of the GP IIB/IIIA Receptors to Avoid Vascular Occlusion (BRAVO)

Sponsor: SmithKline Beecham

Purpose: TIA, Stroke and Heart Attack. The purpose of the Bravo study is to determine the effectiveness of the investigational medication when taken with aspirin in preventing a second stroke, TIA or heart attack.

Investigators: Dr. Norman Gordon, Principal Investigator; Dr. Peter Bellafiore, Sub Investigator; Dr. Vlad Zayas, Sub Investigator

Patients Recruited: Patients who meet the following criteria may be eligible to participate: An ischemic stroke in the last 30 days; A TIA within the last 30 days; A diagnosis of unstable angina or heart attack in the last 14 days.

Intervention: The main benefit is the possibility that the patient's condition may improve or reduce the risk of death, future heart attack, stroke or rehospitalization for vascular disease.

Duration: The study will last from 6 months to up to two years with possible extensions.

Phase: III

Sites: CNS Research, Inc., 100 Butler Drive, Providence, RI 02906. Dr. Peter Bellafiore, 70 Kenyon Avenue, Suite 21 Wakefield, RI 02879.

Contact: Theresa Shaftel, RN, CRC 1-800-847-4332

e-mail: pepple@cns-research.com

Alzheimer's Study: Safety and Efficacy of Olanzapine on Cognitive Symptoms in Subjects with Mild to Moderate Alzheimer's Disease without Psychosis

Sponsor:Eli Lilly and Company

Purpose: The purpose of this study is to test whether Olanzapine will simultaneously benefit cognition, adverse behaviors and disease progression in Alzheimer's Disease.

Investigators: Dr. Norman Gordon, principal investigator; Dr. Vlad Zayas, subinvestigator.

Patients Recruited: Patients 40 years and older with a diagnosis of probable Alzheimer's Disease.

Intervention: Olanzapine may have benefit in treating primary cognitive symptoms of Alzheimer's Disease in addition to behavioral disturbances associated with this condition.

Duration: This is a 6 month study.

Phase: II

Sites: CNS Research, Inc., 100 Butler Drive, Providence, RI 02906

Contact: Patricia Epple, CCRC 1-800-847-4332

e-mail: pepple@cns-research.com

TOPMAT Epilepsy Study: A Randomized, Double-Blind, Parallel Group, Monotherapy Study to Compare the Safety and Efficacy of two doses of Topiramate in the Treatment of Newly Diagnosed or Recurrent Epilepsy

Sponsor: R.W. Johnson

Purpose : Newly Diagnosed or Recurrent Epilepsy: The purpose of this study is to evaluate the effectiveness of Topiramate when used alone for the treatment of epilepsy.

Principal Investigators: Dr. Tom Morgan, Principal Investigator; Dr. Dennis Aumentado, Sub Investigator

Patients Recruited: Individuals who wish to participate in this study must meet the following criteria: 1) are taking only one or no anti-seizure medication; 2) have had no more than 2 documented seizures during the last 3 months; 3) weigh a mininum of 55 pounds.

Intervention: Randomly selected groups of patients will receive two separate target doses of Topiramate.

Duration: The study will last up to one

year with possible extensions
Phase: IIIb
Sites: CNS Research, Inc.; Dr. Thomas Morgan, 54 Jefferson Blvd., Warwick, RI 02888;
Dr. Dennis Aumentado, 219 Cass Avenue, Woonsocket, RI 02895
Contact: Cathy Imondi, CRC (800) 847-4332 x102
e-mail: pepple@cns-research.com

Earlier vs. Later Levodopa in Parkinson's Disease (ELLDOPA)

Sponsor: NIH and the Parkinson Study Group
Purpose: Joseph Friedman, MD, is conducting this trial to compare the effect of early or late treatment of Parkinson's Disease with L-Dopa to answer the question of whether L-Dopa slows or hastens the progression of Parkinson's Disease.
Patients Recruited: Patients must be 30 years or older, must be diagnosed with Parkinson's Disease within the last 2 years. No eldepryl, L-DOPA, amantadine, anticholinergics, antihistamines, antidepressants, or benzodiazepines for previous 30 days.
Intervention: varying doses of Carbidopa/Levodopa vs. placebo
Duration of study: 9 months
Site: Movement Disorder Unit, Memorial Hospital of RI, 111 Brewster St, Pawtucket, RI 02860
Contact: Margaret Lannon, RN, MS phone: (401) 729-3750

The Effect of Pramipexole on Depressive Symptoms in Parkinson's Disease Patients

Sponsor: Pharmacia and Upjohn, Inc.
Purpose: To evaluate the effectiveness of pramipexole on the depression and other non-motor symptoms in patients with Parkinson's Disease
Principal Investigators: Joseph H. Friedman, MD, and Hubert Fernandez, MD
Patients recruited: Men and women with PD, 30 - 85 years old, who have taken no dopamine agonist medications in the past 3 months and are clinically depressed
Intervention: Pramipexole or placebo
Duration of study: 19 weeks
Phase: IV
Site: Movement Disorders Unit, Memorial Hospital of Rhode Island, 111 Brewster Street, Pawtucket, RI 02860
Contact: Margaret Lannon, RN, MS, phone (401) 729-3750

A Multicenter, Randomized, Double-blinded, Placebo-controlled Study of Azithromycin Use in Patients with Documented Coronary Artery Disease

Sponsor: National Heart, Lung and Blood Institute
Purpose: To assess the efficacy of using this antibiotic against Chlamydia pneumoniae and thereby decrease the frequency of subsequent coronary events.
Investigators at Lifespan: Alfred F. Parisi, MD (Principal Investigator) and Ara Sadaniantz, MD (Co-Investigator)
Patients Recruited: Patients who are 1) three months post myocardial infarction with documented cardiac enzymes, 2) three months post revascularization (CABG or angioplasty), 3) Post cardiac catheterization with documentation of >50% stenosis in any coronary artery.
Intervention: Azithromycin 600 mg one dose per week for one year (52 doses) or matching placebo.
Duration: Recruitment of 4,000 patients nationwide, treated for one year; follow-up for mean duration of 3.5 years
Site: The Miriam Hospital, Division of Cardiology, 164 Summit Avenue, Providence, RI 02906
Contact: Ed Mackler, RN, phone: (401) 793-4795; fax: (401) 331-3679; pager: (401) 785-6331; e-mail address: emackler@lifespan.org

Vital Statistics

Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

Edited by Roberta A. Chevoya

Rhode Island Monthly Vital Statistics Report

Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	Dec. 1998	12 Months Ending with Dec. 1998		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	283	3,079	310.9	3,616.0
Malignant Neoplasms	224	2,469	249.3	6,341.0**
Cerebrovascular Diseases	55	596	60.2	838.0
Injuries (Accident/Suicide/Homicide)	32	360	36.4	6,765.0
COPD	41	456	46.1	322.5

Vital Events	Reporting Period		
	June 1999	12 Months Ending with June 1999	
	Number	Number	Rates
Live Births	1,086	13,464	13.6*
Deaths	732	9,709	9.8*
Infant Deaths	(10)	(94)	7.0#
Neonatal deaths	(9)	(73)	5.4#
Marriages	866	7,444	7.5*
Divorces	154	3,083	3.1*
Induced Terminations	470	4,876	362.2#
Spontaneous Fetal Deaths	21	993	73.8#
Under 20 weeks gestation	(15)	(912)	67.7#
20+ weeks gestation	(6)	(81)	6.0#

**Excludes one death of unknown age

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 990,225

(c) Years of Potential Life Lost (YPLL)

Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.

* Rates per 1,000 estimated population
# Rates per 1,000 live births

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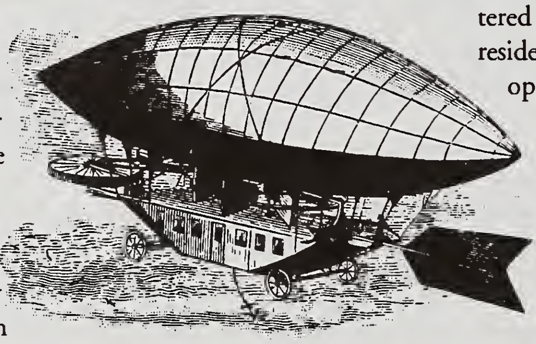
NINETY YEARS AGO

[DECEMBER, 1909]

Charles Chapin, MD, reported 283 deaths for August (15.2 per thousand) and 254 deaths for September (14.5 per thousand - "less than any year for 53 years.") During the past summer, 71 people died from diarrheal diseases. In the past 4 months, he counted 98 cases of scarlet fever, but no deaths - a marked diference from 1896-97, when the case mortality reached 18%

At a regular meeting of the Medical Society, Dr. Harold Palmer read a paper on Clinical Pathology of the Endometrium (with slides shown through the epidiascope), and Dr. W. H. Jordan read a paper on infant feeding.



FIFTY YEARS AGO

[NOVEMBER, 1949]

In "Epidermoid Carcinoma of the Tongue," Drs. Thomas Perry, Jr., and Edward S. Cameron reviewed 95 cases seen in the Rhode Island Hospital Tumor Clinic from 1930 to 1945. Most (89.5%) were male... 24.5% had tested positive on a Wasserman or Hinton test; 15 were Grade 1 tumors, 44 Grade II tumors. Only 15.8% showed a 3-year cure. The authors related the size to the prognosis: "The large size these lesions reach before they are seen in the clinic is appalling...the tendency to consider the tumor an obstinate canker sore has been fatal to many. We are glad to state that only rarely is a physician to blame. Usually the patients do not seek aid of anyone..."

Edward Damaryian, MD, reported on "Anesthesia in Cardiac Arrhythmias and Conduction Disturbances."

Months earlier British journalist Cecil Palmer in a tour of America had addressed the Conference of Presidents and Other Officers of State Medical Societies - an address published in the June 1949 issue of the Journal. This issue included a follow-up article, "The Degradation of British Medicine under Socialism."

TWENTY FIVE YEARS AGO

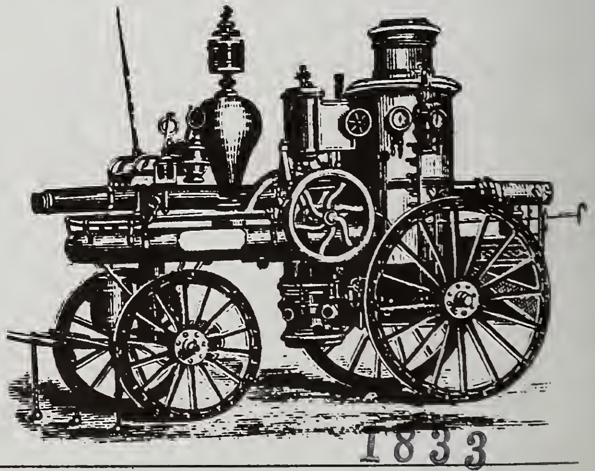
[NOVEMBER, 1974]

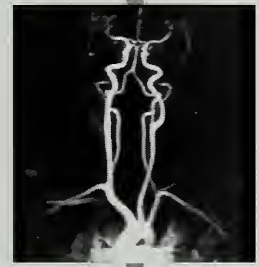
In "Quest for Cure," David S. Greer, MD, Associate Dean of Medical Affairs, described the case of a 24 year-old wheelchair-bound female with multiple sclerosis, admittted to a university hospital in Boston when her mother died. Instead of being discharged to a nursing home, she went to a modern studio apartment in a hospital-connected sheltered housing project in Fall River: "Such patients frequently reside in institutions, victims of social isolation and restricted opportunity as well as neuromuscular deficiency."

The Report of the Committee on Drug Abuse, commenting on the Rhode Island Uniform Control Substances Act of 1974, noted that the Medical Society had lobbied for lenient restrictions (e.g., possession of marijuana was a misdemeanor not a felony; the law distinguished between the seller and possesser of drugs), as well as a 2- year limit on retention of prescription data, "to protect patient rights."

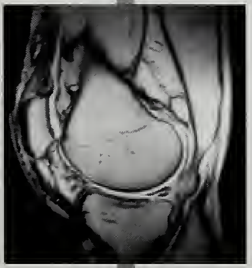
In "The Oral Hypoglycemic Agents in the Treatment of Diabetes Mellitus," Joseph R. Tucci, MD, cautioned, "There is no absolute indication for the use of [these agents]."

Helen E. DeJong, the Medical Society Librarian, argued for retention of the library, in "Si Monumentum Requiris Circumspice" {If you require a monument, look around.] She cited Grace Dickerman's bequest ("modest estate") for the Dickerman Room, which housed 30,000+ volumes. A supporting editorial concurred: "Once our library has been dispersed, it will be like the fallen Humpty-Dumpty." The problem was money; yet the editorial noted that, while an "increase is inevitable," "only 7 state societies have a lower annual dues (by a very few dollars)...A number of them are 50% or higher."





Splenic vein
thrombosis



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